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Volume IV. Benzo[a]pyrene, Acenaphthylene, Benz[a]anthracene, Chrysene, Dibenz[a,h]anthracene, Benzo[b]fluoranthene, Benzo[k]fluoranthene, Benzo[g,h,i]perylene, Indeno[1,2,3-c,d]pyrene

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16. Abstract (Limit: 200 words)

This report assesses the risk of exposure to polycyclic aromatic hydrocarbons (PAHs). This is Volume IV of a four-volume report, analyzing 16 PAHs; it concerns nine of these: benzo[a]pyrene, acenaphthylene, benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[g,h,i]perylene, chrysene, dibenz[a,h]anthracene, and indeno[1,2,3-c,d]pyrene. This study is part of a program to identify the sources of and evaluate exposure to 129 priority pollutants. The analysis is based on available information from government, industry, and technical publications assembled in June of 1981.

The assessment includes an identification of releases to the environment during production, use, or disposal of the substances. In addition, the fate of PAHs in the environment is considered; ambient levels to which various populations of humans and aquatic life are exposed are reported. Exposure levels are estimated and available data on toxicity are presented and interpreted. Information concerning all of these topics is combined in an assessment of the risks of exposure to PAHs for various subpopulations.

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Exposure
Risk
Water Pollution
Air Pollution

Effluents
Waste Disposal
Food Contamination
Toxic Diseases

Polycyclic Aromatic Hydrocarbons
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Benzo[g,h,i]perylene
Benzo[b]fluoranthene
Benzo[k]fluoranthene
Dibenz[a,h]anthracene

Chrysene
Acenaphthylene
PAHs
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AN EXPOSURE AND RISK ASSESSMENT FOR BENZO[a]PYRENE AND OTHER POLYCYCLIC AROMATIC HYDROCARBONS:

VOLUME IV. BENZO[a]PYRENE, ACENAPHTHYLENE, BENZ[a]ANTHRACENE, BENZO[b]FLUORANTHENE, BENZO[k]FLUORANTHENE, BENZO[g,h,i]PERYLENE, CHRYSENE, DIBENZ[a,h]ANTHRACENE, AND INDENO[1,2,3-c,d]PYRENE

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OFFICE OF WATER REGULATIONS AND STANDARDS
OFFICE OF WATER
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WASHINGTON, D.C. 20460

FOREWORD

Effective regulatory action for toxic chemicals requires an understanding of the human and environmental risks associated with the manufacture, use, and disposal of the chemical. Assessment of risk requires a scientific judgment about the probability of harm to the environment resulting from known or potential environmental concentrations. The risk assessment process integrates health effects data (e.g., carcinogenicity, teratogenicity) with information on exposure. The components of exposure include an evaluation of the sources of the chemical, exposure pathways, ambient levels, and an identification of exposed populations including humans and aquatic life.

This assessment was performed as part of a program to determine the environmental risks associated with current use and disposal patterns for 65 chemicals and classes of chemicals (expanded to 129 "priority pollutants") named in the 1977 Clean Water Act. It includes an assessment of risk for humans and aquatic life and is intended to serve as a technical basis for developing the most appropriate and effective strategy for mitigating these risks.

This document is a contractors' final report. It has been extensively reviewed by the individual contractors and by the EPA at several stages of completion. Each chapter of the draft was reviewed by members of the authoring contractor's senior technical staff (e.g., toxicologists, environmental scientists) who had not previously been directly involved in the work. These individuals were selected by management to be the technical peers of the chapter authors. The chapters were comprehensively checked for uniformity in quality and content by the contractor's editorial team, which also was responsible for the production of the final report. The contractor's senior project management subsequently reviewed the final report in its entirety.

At EPA a senior staff member was responsible for guiding the contractors, reviewing the manuscripts, and soliciting comments, where appropriate, from related programs within EPA (e.g., Office of Toxic Substances, Research and Development, Air Programs, Solid and Hazardous Waste, etc.). A complete draft was summarized by the assigned EPA staff member and reviewed for technical and policy implications with the Office Director (formerly the Deputy Assistant Administrator) of Water Regulations and Standards. Subsequent revisions were included in the final report.

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Exposure Assessment Section
Monitoring & Data Support Division (WH-553)
Office of Water Regulations and Standards

AN EXPOSURE AND RISK ASSESSMENT FOR BENZO[a]PYRENE AND OTHER POLYCYCLIC AROMATIC HYDROCARBONS

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1.0 Introduction
2.0 Technical Summary

VOLUME II Naphthalene

3.0

3.1 Materials Balance
3.2 Fate and Distribution in the Environment
3.3 Effects and Exposure--Humans
3.4 Effects and Exposure--Aquatic Biota
3.5 Risk Considerations

VOLUME III Anthracene, Acenaphthene, Fluoranthene, Fluorene, Phenanthrene, and Pyrene

4.0

4.1 Materials Balance
4.2 Fate and Distribution in the Environment
4.3 Effects and Exposure--Humans
4.4 Effects and Exposure--Aquatic Biota
4.5 Risk Considerations

VOLUME IV Benzo[a]pyrene, Acenaphthylene, Benz[a]anthracene, Benzo[b]fluoranthene, Benzo[k]fluoranthene, Benzo[g,h,i]perylene, Chrysene, Dibenz[a,h]anthracene, and Indeno[1,2,3-c,d]pyrene

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The Materials Balance for the BaP group PAHs (Section 3.1) was produced by Acurex Corporation, under Contract No. 68-01-6017 to the Monitoring and Data Support Division (MDSD), Office of Water Regulations and Standards (OWRS), U. S. Environmental Protection Agency. Patricia Cruse was the task manager for Acurex, Inc.; other contributors include Robert DeRosier, Kenneth Moss and Stephen Wendt. Patricia Leslie was responsible for report production on behalf of Acurex, Inc.

John Segna and Michael Slimak were the EPA project managers for this assignment.

1.0 INTRODUCTION

The Office of Water Regulations and Standards (OWRS), Monitoring and Data Support Division, of the U.S. Environmental Protection Agency is conducting a program to evaluate the exposure to and risk of 129 priority pollutants in the nation's environment. The risks to be evaluated include potential harm to human beings and deleterious effects on fish and other biota. The goals of the program under which this report has been prepared are to integrate information on cultural and environmental flows of specific priority pollutants, to estimate the likelihood of receptor exposure to these substances, and to evaluate the risk resulting from such exposures. The results are intended to serve as a basis for estimating the magnitude of the potential risk and developing a suitable regulatory strategy for reducing any such risk.

This report, comprised of four separate volumes, provides a summary of the available information concerning the releases, fate, distribution, effects, exposure, and potential risks of the 16 priority pollutants that are polycyclic aromatic hydrocarbons (PAHs). The chemical structures of these compounds are shown in Figure 1-1.

The number of chemicals considered in this exposure and risk assessment is appreciable. The possibility of preparing 16 separate exposure and risk assessment documents was considered and rejected because it would lead to considerable redundancy and because so little information was available on some of the individual PAHs. As an alternative, the 16 PAHs were organized at the onset of the work into three groups, as indicated in Figure 1-1.

The rationale for the organization into these three specific groups included considerations of materials balance, chemical properties related to fate and environmental pathways, and health effects, as described briefly below.

- Naphthalene is the only one of the 16 PAHs with substantial U.S. commercial production and with a significant potential for direct exposure to consumers of a commercial product (mothballs). It is significantly more volatile and more water soluble than any other PAH. It was not anticipated to have carcinogenic effects in humans.

- Anthracene, acenaphthene, fluorene, fluoranthene, phenanthrene and pyrene are all imported in rather small quantities for special commercial uses. These compounds are three- and four-ring PAHs, with moderately low volatility and water solubility. The question of their possible carcinogenicity was expected to require careful review. Most of the information pertaining to this group is specific to anthracene.

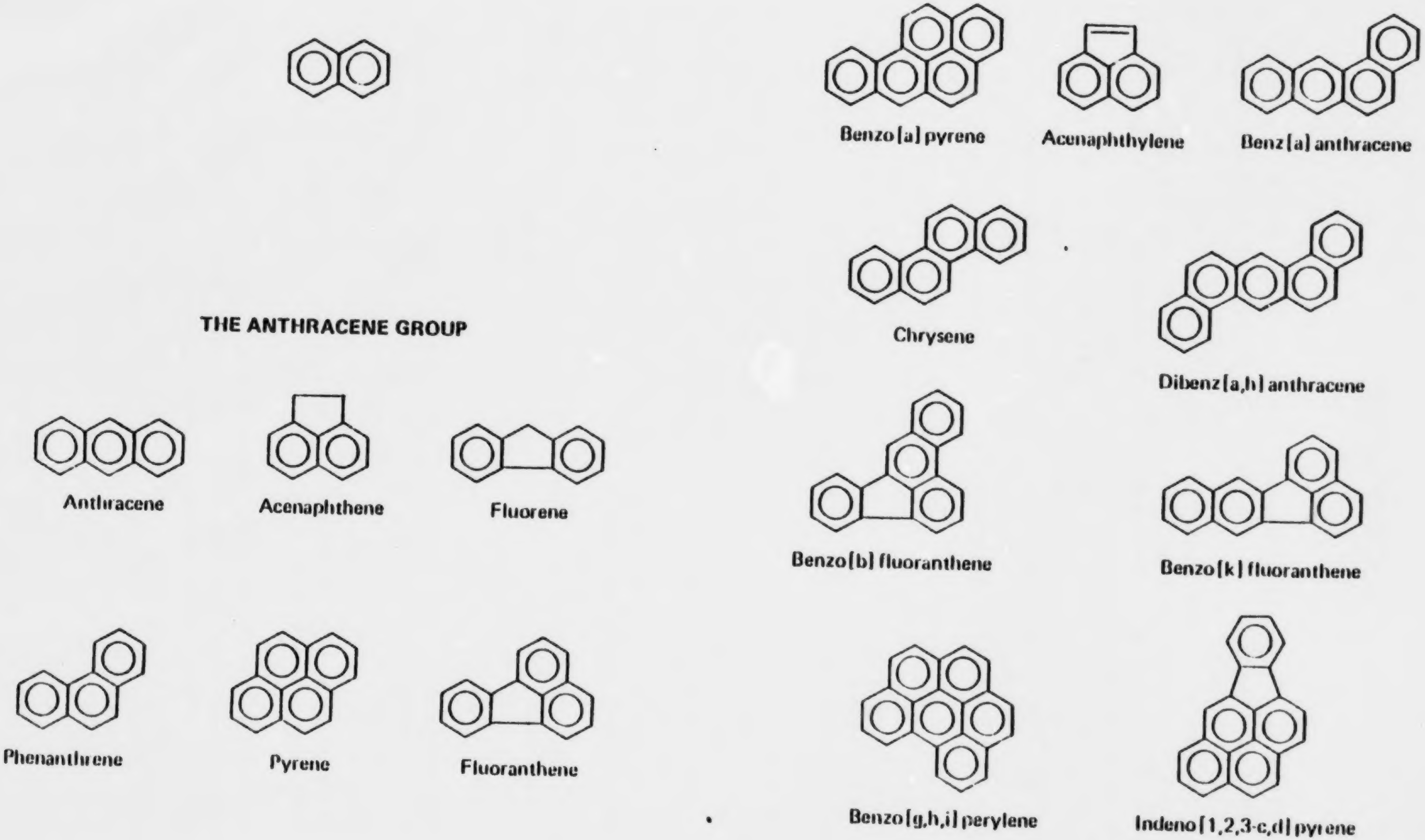
Benzo [a] pyrene
Acenaphthylene
Benz [a] anthracene
Chrysene
Dibenz [a,h] anthracene
THE ANTHRACENE GROUP
Anthracene
Acenaphthene
Fluorene
Benzo [b] fluoranthene
Benzo [k] fluoranthene
Phenanthrene
Pyrene
Fluoranthene
Benzo [g,h,i] perylene
Indeno [1,2,3-c,d] pyrene

FIGURE 1-1 STRUCTURES OF THE PRIORITY POLLUTANT POLYCYCLIC AROMATIC HYDROCARBONS

- Benzo[a]pyrene (BaP) and the eight other PAHs in the third group have no commercial production or use, except as research laboratory standards. They are released to the environment inadvertently by combustion sources. With one exception (acenaphthylene), the chemicals in this group have very low vapor pressures and water solubilities. Several of the PAHs in the BaP group had been identified as carcinogens. Much of the information regarding this group of compounds is for BaP.

The exposure and risk assessment for each of the three groups of PAHs was treated in a separate chapter of a multivolume report; Chapter 3.0 (Volume II) concerns naphthalene; Chapter 4.0 (Volume III) concerns the anthracene group PAHs; and Chapter 5.0 (Volume IV) concerns the benzo[a]pyrene group PAHs. These chapters are bound separately.

Potential waterborne routes of exposure are the primary focus of these exposure and risk assessments because of the emphasis of OWRS on aquatic and water-related pathways. Inhalation exposures are also considered, however, in order to place the water-related exposures into perspective. Each chapter contains major sections discussing the following topics:

- Information on environmental releases of the subject PAHs, including the form and amounts released and the receiving medium at the point of entry into the environment (materials balance);
- Description of the fate processes that transform and/or transport the compounds from the point of release through environmental media until exposure of humans and other receptors occurs, and a summary of reported concentrations detected in the environment, with a particular emphasis on aquatic media;
- Discussion of the available data concerning adverse health effects of the subject PAHs on humans, including (where known) the doses eliciting those effects and an assessment of the likely pathways and levels of human exposure;
- Review of available data concerning adverse effects on aquatic biota and the levels of environmental exposure; and
- Discussion of risk considerations for various subpopulations of humans and other biota.

Two comments regarding the materials balance section are appropriate. First, these sections were based in large part on draft material prepared by Acurex Corporation, under EPA Contract 68-01-6017, and provided to Arthur D. Little, Inc. by EPA. Second, the phrase "materials balance" is somewhat inappropriate when applied to chemicals such as the PAHs that are produced primarily as byproducts of combustion processes. Since most PAH production is inadvertent rather than deliberate commercial production, the conventional approach of trying to balance production versus use and environmental release is not strictly applicable to these chemicals. Therefore, the materials balance sections of these exposure and risk assessments are focused on estimates of releases from major sources such as combustion; considerable uncertainty is associated with most of these estimates.

After an initial review of the three exposure and risk assessments covering all 16 priority pollutant PAHs, it was determined that one chemical, benzo[a]pyrene, was of appreciably greater interest to OWRS than were the other 15 compounds studied. This interest reflects the more extensive data base available for assessment of environmental fate and exposure and also the existence of some, although limited, dose-response data to which various extrapolation models can be applied for estimation of potential human carcinogenic risk from ingestion of BaP. For the other PAHs considered, data on carcinogenic or other long-term effects were generally limited, nonquantitative, and/or did not indicate statistically positive results. Table 1-1 presents a summary of the hazard of the 16 priority pollutant PAHs in terms of carcinogenicity, based on qualitative review of available information.

For these reasons, the technical summary presented in Volume I is organized somewhat differently than the rest of the report (Chapter 3.0-5.0) (Volumes II-IV). The summary is focused on benzo[a]pyrene as the PAH of greatest interest. The estimated releases to the environment, environmental fate, monitoring data, human effects and exposure, biotic effects and exposure, and risk considerations concerning BaP are presented in expanded summary form. Abbreviated summaries are then provided for naphthalene, anthracene group PAHs, and the other PAHs considered.

Included in the summary volume are critical data and references to the literature so that this volume may be read and understood by itself without reference to the separately bound Chapters 3.0-5-0. The latter volumes contain more extensive compilations of data, more detailed discussions of the available information and of the interpretations drawn, and more complete documentation of the multiple literature sources that were reviewed in the course of this work.

TABLE 1-1. SUMMARY OF EVIDENCE FOR CARCINOGENICITY OF PRIORITY POLLUTANT PAHs

PAH*	Basis
Benzo[a]pyrene	Positive oral carcinogen with other positive carcinogenic data.
Dibenz[a,h]anthracene	Positive oral carcinogen with other positive carcinogenic data.
Benz[a]anthracene	Positive oral carcinogen with other positive carcinogenic data.
Benzo[g,h,i]perylene	Not tested orally, other positive carcinogenic or co-carcinogenic data.
Benzo[b]fluoranthene	Not tested orally, other positive carcinogenic or co-carcinogenic data.
Chrysene	Not tested orally, other positive carcinogenic or co-carcinogenic data.
Indeno[1,2,3-c,d]pyrene	Co-carcinogen or initiator with negative carcinogen or *in vivo* mutagen.
Pyrene	Co-carcinogen or initiator with negative carcinogen or *in vivo* mutagen.
Fluoranthene	Co-carcinogen or initiator with negative carcinogen or *in vivo* mutagen.
Benzo[k]fluoranthene	Negative in a single carcino genic study.
Phenanthrene	Several negative carcinogenic and mutagenic studies but not tested orally.

TABLE 1-1. SUMMARY OF EVIDENCE FOR CARCINOGENICTY OF PRIORITY POLLUTANT PAHs (Continued)

Anthracene	Negative studies, tested orally.
Naphthalene	Negative studies, tested orally.

*No data for evaluation of carcinogenicity were available for acenaphthene, acenaphthylene, or fluorene.

5.0 BENZO[a]PYRENE, ACENAPHTHYLENE, BENZ[a]ANTHRACENE, BENZO[b]FLUORANTHENE, BENZO[k]FLUORANTHENE, BENZO[g,h,i]PERYLENE, CHRYSENE, DIBENZ[a,h]ANTHRACENE, IDENO[1,2,3-c,d]PYRENE

5.1 MATERIALS BALANCE

5.1.1 Introduction

This section reviews both published and unpublished data concerning the production, use, and disposal of the benzo[a]pyrene (BaP) group PAHs in the United States. Information from the available literature has been reviewed to present an overview of major sources of environmental releases of these compounds. Annotated tables have been included to aid data evaluation.

The section is organized according to the major categories of the sources of releases to the environment. Section 5.1.2 describes the numerous combustion processes that release the BaP group PAHs; Section 5.1.3 discusses contained sources; and Section 5.1.4 describes the amounts of these compounds in the influents and effluents of Publicly Owned Treatment Works (POTWs). Section 5.1.5 is a summary of the materials balance.

5.1.2 Combustion Sources

Combustion is the major source of PAH releases to the environment. This section estimates releases of BaP group PAHs from residential heating, fireplaces, cigarettes, coal refuse piles, wildfire, carbon black manufacture, gasoline use, and utility boilers; the emission estimates are summarized in Table 5-1.

5.1.2.1 Residential Coal Combustion

Residential coal combustion is responsible for a significant fraction (5%) of PAH emissions for the compounds in this group. Since residential heating units are typically not controlled and are relatively inefficient, they produce large amounts of PAHs. However, the trend in energy utilization for home heating sources suggests a rapidly decreasing reliance on coal. Whereas coal was used for heating in 2.7% of residences in 1970, it was used in only 0.56% in 1977 (Census 1979). (This trend may have reversed in more recent years.) Figure 5-1 shows the estimated distribution of residential coal consumption by state. Emissions from residential coal combustion (see Table 5-1) were estimated from the emission factors in Appendix A, Table A-1 (EPA 1977a) and the 1978 coal consumption of 8688 x 10^3 kkg for residential heating use (DOE 1980).

Table 5-1. Estimated Air Emission of Benzo[a]pyrene-Group PAHs by Combustion Source, 1978 (kkg)

	Residential Coal Combustion	Fireplaces	Primary Residential Wood Heating	Auxiliary Residential Wood Heating	Cigarettes	Coal Refuse Piles	Prescribed Burning	Wildfire	Carbon Black	Tire Wear[b]	Agricultural Burning	Gasoline	Utility Boilers Coal	Utility Boilers Oil	Incinerators	Utility and Industrial IC Engines	Gas- and Oil- Fired Residential Sources
Acenaphthylene		30	400	500					1							1	
Benzo[a]anthracene	20	2	50	70	neg[a]		100	60	neg		40				neg		
Benzo[b]fluoranthene	20	2	40	50		neg	50	30	neg	neg	20				neg		
Benzo[b]fluoranthene	20	2	40	50		neg	50	30	neg	neg	20				neg		
Benzo[ghi]perylene	-	3	40	50	neg		90	50	neg	8	30	40	1	1	neg		
Benzo[a]pyrene	10	2	30	40	neg	neg	30	20	neg	1	10	10	1	1	neg		
Chrysene	20	2	50	70			100	60	neg		40				neg		
Dibenzo[a,h]anthracene	30	neg	5	6		neg											
Indeno[a,2,3-cd]pyrene	20		neg	neg	neg	neg	60	30	neg	1	20				neg		
Total	140	43	655	836			400	200	1	10	180	50	2	2		1	neg

a) Negligible defined <1 kkg/year. Blanks indicate data not available. Emissions factors are presented in Table A-1, Appendix A. The text and Appendix A describe derivations of estimates.

b) PAH strongly bound to particulate carbon blank.

Sources: See text.

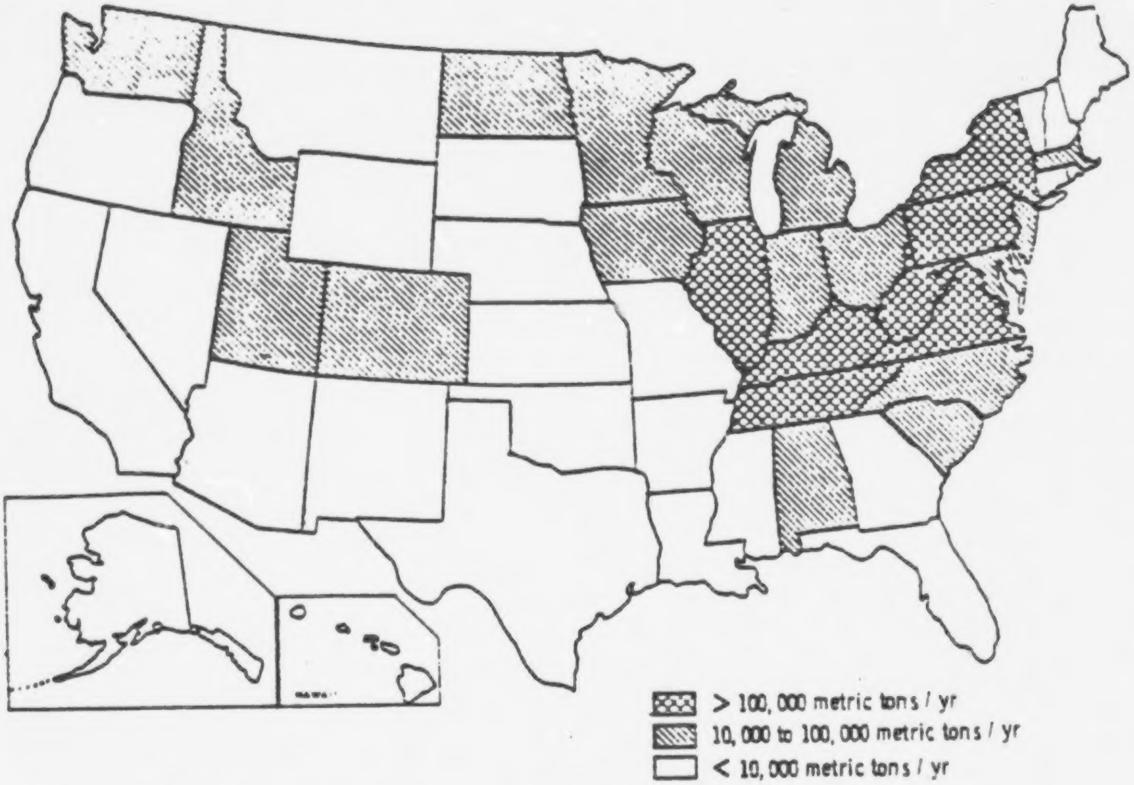
> 100,000 metric tons / yr
10,000 to 100,000 metric tons / yr
< 10,000 metric tons / yr

Figure 5-1. Estimated Residential Coal Consumption in 1974 by State (EPA 1977a)

5.1.2.2 Wood

Wood may be burned in homes in fireplaces or in stoves or furnaces for primary or for auxiliary heating. Estimated PAH emissions from these sources are listed in Table 5-1. Fireplaces produce smaller amounts of PAHs per kg of fuel than does wood combustion in primary heating units; in addition, fireplaces use less wood overall than is consumed for heating. The amounts of wood burned in fireplaces, for primary heating and for auxiliary heating in 1977, were 2.9×10^6 kkg, 6.9×10^6 kkg, and 9.2×10^6 kkg, respectively (see Appendix A, Note 1). Table 5-2 lists the estimated 1976 wood consumption for residential burning by state (EPA 1980a). The number of homes heated by wood has increased by more than 50%, from 794,000 in 1970 to 1,239,000 in 1977 (Census 1979).

Emissions of BaP group PAHs from these sources were calculated using the emission factors presented in Table A-1 (Appendix A) and 1977 wood consumption of 6.9×10^6 kkg and 9.2×10^6 kkg for primary and auxiliary heating, respectively. The emission factors presented in Table A-1 are averages of those for baffled and nonbaffled wood stoves (EPA 1980a). Total 1977 wood consumption for primary heating was obtained from the number of housing units that used wood for primary heat in 1976 and 1977 (912,000 and 1,239,000) and a proportional extrapolation of the estimated 5.1×10^6 kkg of wood burned in 1976 to 6.9×10^6 kkg for 1977 (EPA 1980a and Census 1979). Total 1977 wood consumption for auxiliary heating (9.2×10^6 kkg) was extrapolated from the 1976 estimate of 8.5×10^6 kkg (EPA 1980a) on the basis of the increase in the number of houses with fireplaces (Appendix A, Note 1). The estimated wood consumption for each state is shown in Table 5-2.

5.1.2.3 Cigarettes

Although cigarette smoking may be the source of a significant PAH exposure to individuals, it is a small contributor to national PAH emissions. Cigarette smoking contributes <1 kkg/yr of each of the BaP group PAHs to the atmosphere.

The emission factors for cigarette smoking, shown in Table A-1, were used, along with the number of cigarettes produced (616×10^9) (USDA 1979) in order to determine the amounts of BaP group PAHs emitted. The emission factors for the BaP group compounds are all from Neff (1979).

5.1.2.4 Coal Refuse Piles

Coal refuse piles, impoundments, abandoned mines, and outcrops may burn through the spontaneous combustion of coal. Because of the relatively poor air supply and uneven heat distribution, this combustion is inefficient and, therefore, produces relatively large amounts of PAHs. The estimated 52×10^6 kkg of coal in refuse piles (EPA 1978a) produce

Table 5-2. Estimated Residential Wood Consumption by State, 1976 (kkg)

State	Primary heating	Fireplaces	Auxiliary Wood Stoves
Alabama	150,000	34,000	360,000
Alaska	50,000	5,000	32,000
Arizona	36,000	34,000	26,000
Arkansas	280,000	21,000	100,000
California	190,000	360,000	270,000
Colorado	18,000	40,000	61,000
Connecticut	22,000	29,000	140,000
Delaware	5,000	5,000	13,000
Florida	30,000	88,000	210,000
Georgia	340,000	46,000	220,000
Hawaii	1,000	12,000	9,000
Idaho	60,000	12,000	19,000
Illinois	29,000	120,000	260,000
Indiana	58,000	58,000	120,000
Iowa	16,000	32,000	68,000
Kansas	24,000	26,000	56,000
Kentucky	190,000	32,000	170,000
Louisiana	38,000	35,000	84,000
Maine	180,000	16,000	440,000
Maryland	47,000	39,000	94,000
Massachusetts	26,000	91,000	270,000
Michigan	76,000	97,000	410,000
Minnesota	110,000	43,000	360,000
Mississippi	200,000	21,000	110,000
Missouri	330,000	55,000	230,000
Montana	60,000	12,000	18,000
Nebraska	12,000	18,000	39,000
Nevada	15,000	10,000	8,000
New Hampshire	57,000	13,000	340,000
New Jersey	19,000	114,000	170,000
New Mexico	120,000	69,000	25,000
New York	150,000	290,000	870,000
North Carolina	340,000	51,000	120,000
North Dakota	3,000	7,000	14,000
Ohio	47,000	120,000	250,000
Oklahoma	78,000	28,000	34,000
Oregon	290,000	38,000	250,000
Pennsylvania	120,000	190,000	280,000
Rhode Island	5,000	15,000	22,000
SouthCarolina	190,000	26,000	62,000
South Dakota	20,000	7,000	16,000
Tennessee	320,000	41,000	850,000
Texas	87,000	120,000	290,000
Utah	11,000	18,000	25,000
Vermont	33,000	7,000	190,000
Virginia	280,000	48,000	110,000
Washington	210,000	58,000	390,000
West Virginia	36,000	18,000	43,000
Wisconsin	91,000	50,000	210,000
Wyoming	10,000	6,000	9,000
Total	5,100,000	2,700,000	8,800,000

Source: EPA 1980a

the estimated PAH emissions shown in Table 5-1. The emissions do not account for burning refuse in impoundments or abandoned mines and outcrops since the quantity of burning coal is impossible to measure or estimate (EPA 1978a). It is, however, estimated that only 21% of the mass present in coal refuse piles is burning at any given time (EPA 1978a). Table 5-3 presents a distribution of burning coal refuse by state. It is worth noting that Pennsylvania and West Virginia account for 80% of these fires. Although the emissions were not estimated, the states of Montana, Wyoming, Colorado, and New Mexico account for 66% of the approximately 400 burning abandoned mines or outcrops.

From the composition of the particulate polycyclic organic matter (POM) shown in Table A-1, emissions were calculated based upon a refuse pile volume of 190 x 10^6 m^3 (21% of which is estimated to be burning), a density of 1.5 kkg/m^3, and a POM emission rate of 1.3 x 10^{-8} kg/kkg-hr (EPA 1978a). Only particulate emissions were analyzed here; and only preliminary sampling data are presented. Based upon these assumptions, the emissions are estimated to be less than 1 kkg. Alternatively, NAS (1972) gives an unsubstantiated estimate of 340 tons/year of BaP from coal refuse piles.

5.1.2.5 Forest Fires

Forest fires, either intentional or not (wildfire), are a major source of PAHs. Unfortunately, the estimates of PAHs produced are based on data that do not adequately cover the range of situations that may be termed wildfire. PAH production may be very crudely determined by using estimates of the amount of fuel burned and an emission factor. The amount of fuel burned (in terms of both fuel per unit area and total area burned) is for the most part based upon experienced judgments (EPA 1979a). Emissions factors are based upon laboratory simulations, which do not test the wide range of fuel type and configurations and may not appropriately simulate the fire intensity, weather conditions, fuel moisture, etc.

Prescribed burning is a controlled form of open burning used in land management to achieve specific objectives, which include fire hazard reduction, disease control, and silviculture, among others. An estimated 36 x 10^6 kkg (dry weight) of fuel are burned annually, not including agricultural open burning (Pierovich 1978)(see Section 5.1.2.7). Table 5-4 presents a breakdown of this fuel usage by region; the estimated emissions for this source are presented in Table 5-1. These estimates were calculated from the amount of fuel burned and the emission factors for forest fires shown in Table A-1 (McMahon and Tsoukalas 1977).

Table 5-3. Coal Refuse Fires

State	Amount of Coal Refuse (10^3 m^3)
Alabama	11,000
Colorado	12,000
Illinois	5,000
Kentucky	1,600
Maryland	23
Montana	230
Ohio	1,400
Pennsylvania	84,000
Utah	2,100
Virginia	2,800
Washington	2,300
West Virginia	67,000
Total[a]	190,000

a) Total does not add due to independent rounding

Source: EPA 1978a

Table 5-4. Estimated Net Total Weights of Fuel Consumed Annually in Prescription Burning on All Ownerships

Geographic region[a]	Net annual consumption, 10^5 metric tons (dry weight)			
	Timber harvesting and land clearing residues[b]		Naturally occurring understory vegetation and litter	Total of all categories
	Piles or windrows	Broadcast (unpiled)		
Alaska	0.5			0.5
Eastern	0.1	0.3	0.1	0.5
Intermountain	6.9	0.7		7.6
Northern	82.7	6.3		89.0
Pacific Northwest	50.4	37.0		87.4
Pacific Southwest	20.2	8.2	0.5	28.9
Rocky Mountain	4.9	1.0	0.2	6.1
Southern	0.9	9.9	114.2	125.0
Southwestern	12.3	8.3	0.3	20.9
Total of all regions	178.9	71.7	115.3	365.9

a) See Figure 5-2 for states included in each region

b) Includes precommercial thinning residues as follows: Pacific Southwest and Rocky Mountain, each <0.1 x 10^5 metric tons; Southwestern, 1.1 x 10^5 metric tons.

Source: Pierovich 1978.

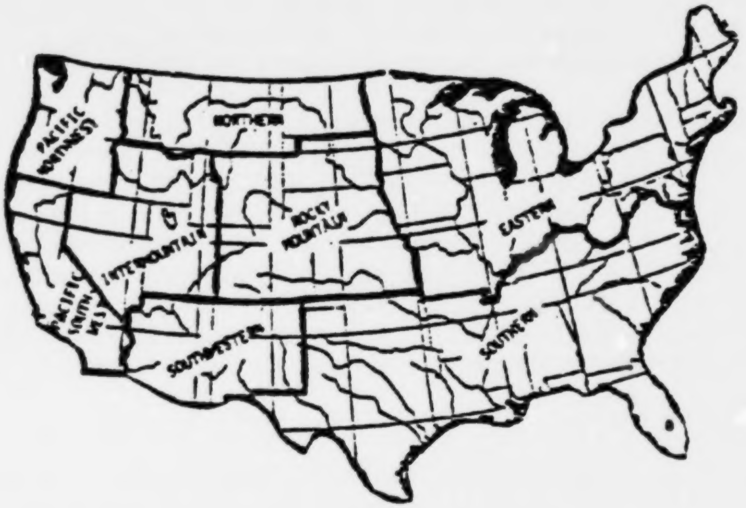
PACIFIC NORTHWEST
NORTHERN
ROCKY MOUNTAIN
INTERMOUNTAIN
PACIFIC SOUTHWEST
SOUTHWESTERN
EASTERN
SOUTHERN

Figure 5-2. Geographic Regions used to Summarize Survey Data Within the Coterminous States. (Alaska Region also used in summarization not shown.)

Source: Pierovich 1978

Land burned by wildfire each year varies considerably in both the total area burned and the location of specific sites. On the average, 1 x 10^{10} m^2 (3 x 10^6 acres) are burned each year, although in 1976, 2 x 10^{10} m^2 (5 x 10^6 acres) were burned (Dahl 1980). The largest area is burned in the Southeastern section of the country, followed by the South, North Central, Pacific, and Rocky Mountain sections (Dahl 1980). Emissions estimates are shown in Table 5-1.

The emissions factors for prescribed burning and wildfire in Table A-1 (McMahon and Tsoukalas 1977) are averages obtained from six tests on pine needles, with differing fuel densities used for heading and backing fires; hence, these factors cannot provide a very accurate basis for nationwide emissions estimates. The total amount of fuel burned was estimated by assuming that 10^{10} m^2 (3 x 10^6 acres) of land were burned on the average (Dahl 1980), and that the fuel loading was 2 kg consumed dry weight/m^2 (based upon estimates by EPA 1978b). Total quantities of BaP group PAHs emitted from this source are estimated in Table 5-1.

5.1.2.6 Carbon Black

Carbon black is produced primarily by the furnace process, in which a liquid hydrocarbon feedstock is incompletely pyrolyzed in a refractory-lined furnace by burning natural gas. The carbon black particles are recovered in a baghouse after being cooled by water sprays. The carbon black is then converted to a marketable product. The principal emission source from carbon black manufacture is baghouse exhaust gas from the main process vent. PAHs are present both in the vent gas and on the carbon black itself (see Appendix A, Note 2 and Tables A-1 and A-3). For the latter reason, carbon black was banned in food, drugs, and cosmetics in 1976.

Emissions of PAHs from carbon black manufacture are presented in Table 5-1; Table 5-5 lists the location and capacity of carbon black producers. For the most part, the emissions during production are negligible (i.e., <1 kkg/yr). Larger amounts, however, are contained in/on the carbon black. These are shown in Table A-3.

The primary use for carbon black is in the elastomers used in automobile tires (65%), while other elastomeric uses account for 29% (SRI 1979). The PAHs present on the carbon black are strongly bound to it and, therefore, are not likely to be easily separated from it, except by prolonged extraction (Locati et al. 1979). An estimate of PAH air emissions from tire wear, presumably associated with carbon black, is discussed in Note 2 and presented in Table 5-1 (see Appendix A). PAHs associated with carbon black that is transferred to road surfaces are presented in Table A-4. Although this release presents a possible source of PAHs in runoff, these PAHs are probably tightly bound to the carbon black particles .

Table 5-5. Locations and Capacities of Carbon Black Producers

Company and Location	Annual Capacity 10^3 kkg
ASHLAND OIL, INC.	
Ashland Chemical Company, division	
Carbon Black and Synthetic Rubber Division	
Aransas Pass, Texas	74
Belpre, Ohio	45
Iberia, Louisiana	120
Mojave, California	30
Shamrock, Texas	50
Total	320
CABOT CORPORATION	
Big Spring, Texas	110
Franklin, Louisiana	100
Rampa, Texas	24
Villa Platte, Louisiana	110
Waverly, West Virginia	74
Total	420
CITIES SERVICE COMPANY	
Chemicals Group	
Columbian Chemicals, division	
Conroe, Texas	44
El Dorado, Arkansas	7
Eola, Louisiana	32
Mojave, California	24
Moundsville, West Virginia	71
North Bend, Louisiana	71
North Bend, Louisiana	25
Seagraves, Texas	40
Ulysses, Kansas	23
Total	370
CONTINENTAL CARBON COMPANY (owned by Continental Oil Company 80% and Witco Chemical Corporation, 0%)	
Bakersfield, California	35
Phenix City, Alabama	23
Ponca City, Oklahoma	61
Sunray, Texas	43
Westlake, Louisiana	55
Total	217
J.M. HUBER CORPORATION	
Baytown, Texas	117
Borger, Texas	62
Borger, Texas	19
Total	198
PHILLIPS PETROLEUM COMPANY	
Rubber Chemicals Division	
Borger, Texas	130
Orange, Texas	61
Toledo, Ohio	39
Total	230
SID RICHARDSON CARBON COMPANY	
Addis, Louisiana	46
Big Spring, Texas	54
Total	96
TOTAL	1,850

Totals may not add due to rounding
Source: SRI 1980.

5.1.2.7 Agricultural Open Burning

Agricultural open burning involves burning of crop residues for residue removal, field sanitation, or preparation of farmlands for cultivation. Wastes that may be burned include a wide variety of residues, including rice straw, orchard prunings, potato vines, and bagasse, among others. Although PAH formation is dependent upon fuel type, among the many other variables, the emissions in Table 5-1 were calculated based upon wood burning (forest fire; see Table A-1, Appendix A), and an estimated total of 13 x 10^6 kkg dry weight of burned material (EPA 1977b). Table 5-6 lists the estimated distribution of open burning by state.

5.1.2.8 Motor Vehicles

Motor vehicles that burn gasoline are ubiquitous sources of PAHs, although emissions control devices for hydrocarbons in exhaust gas and from the crankcase reduce the amount emitted at efficiencies up to 99% (Gross 1972). PAHs released by motor vehicles are either emitted in the exhaust or dumped along with the used crankcase oil. Estimated PAH emissions from exhaust gas are presented in Table 5-1 (see Appendix A, Note 3). Although crankcase controls reduce hydrocarbon emissions from the vehicle, PAHs still reside in the crankcase oil and may be released to either land or water if the oil is disposed of haphazardly. Table 5-7 presents estimates of the PAHs contained in these releases. (See Appendix A, Note 3 and Table A-5.)

5.1.2.9 Incinerators

PAH releases from municipal and commercial incinerators are estimated to be negligible (<1 kkg/yr). Releases were calculated for municipal incinerators using the release factors in Table A-6 (Davies 1976) for 104 plants with an average capacity of 385 kkg/day when operating at full capacity (EPA 1978b).

Releases for commercial incinerators were assumed to be similar to those from municipal incinerators. Even when the higher emission factors in Table A-6 are used, calculated emissions of any given PAH were negligible without controls. The incinerators were assumed to number 100,000 units, firing 3 hours/day, 260 days/year at an average capacity of 0.1 kkg/hr (EPA 1978b).

Table 5-6. Distribution of Agricultural Open Burning, 1973

State	Area burned 10^7 m^2/yr	Amount of crop residue burned 10^3 kkg/yr
Alabama	36.	161
Arizona	4.5	20
Arkansas	26.	115
California	309.	2,075
Colorado	32.	143
Delaware	0.1	2
Florida	109.	1,716
Georgia	39.5	883
Hawaii	44.7	1,175
Idaho	4.8	22
Kansas	243.	544
Kentucky	13.	58
Louisiana	142.	1,904
Maine	15.	33
Maryland	0.6	7
Massachusetts	0.6	3
Michigan	21.	93
Minnesota	61.2	274
Mississippi	138.	617
Missouri	40.5	181
Montana	34.	152
Nebraska	67.6	303
Nevada	0.8	5
New Mexico	0.5	2
North Carolina	138.	619
North Dakota	97.	438
Ohio	32.	142
Oklahoma	36.	161
Oregon	107.	479
Pennsylvania	15.	69
South Carolina	8.	38
South Dakota	59.	265
Tennessee	14.	61
Virginia	9.5	43
Washington	57.	256
Wisconsin	32.	145
Wyoming	7.3	34
National Total	1995.	13,238

Source: EPA 1977b.

Table 5-7. BaP Group PAHs Discharged Annually in Used Crankcase Oil[a] (kkg)

PAH	Discharge
Benzo[a]anthracene	2
Benzo[k]fluoranthene	3
Benzo[ghi]perylene	3
Benzo[a]pyrene	neg[b]
Chrysene	2

a) Releases go presumably to POTWs and landfills. No recycling is assumed.

b) Negligible is <1 kkg.

Source: Peake and Parker 1980 and Tanacredi 1977.
See Note 3, Appendix A.

5.1.2.10 Coal- and Oil-fired Utility Boilers

Table 5-1 summarizes PAH emissions from coal- and oil-fired utility boiler units. The estimates of total PAHs released to the environment are based upon the emission factors averaged from Table A -7 and a total coal and oil consumption for electricity generation of 4.8×10^8 and 7.8×10^7 kkg per year, respectively (Monthly Energy Review 1980).

These emissions are uncontrolled releases calculated from 1967 emissions factors, and are probably lower today with the use of baghouses or electrostatic precipitator units. Further, the emission factors for oil are for small- or intermediate-sized units, and thus serve as an upper limit of the PAH releases from higher capacity plants.

5.1.2.11 Utility and Industrial Internal Combustion Engines

Stationary internal combusion engines are used for electricity generation, oil and gas transmission, natural gas processing, and oil and gas production and exploration. These engines are either gas turbines or reciprocating engines and are fueled with either oil or gas. Table 5-1 presents the estimated amount of PAHs emitted from these sources in 1978 as estimated by EPA (1979d).

5.1.2.12 Gas- and Oil-fired Residential Heating

Table 5-1 lists PAH emissions from oil-fired residential heating units as estimated by EPA (1979e). Although a large fraction of residences provide space heat with gas, gas-fired units emit negligible amounts of PAHs. Figure 5-3 presents the geographic distribution of oil and gas used for residential heating in 1975 (EPA 1979e).

5.1.3 Contained Sources

This section presents information, primarily in tabular format, on sources of BaP group PAHs from which these compounds are not specifically isolated. Delineation of all such "inadvertent sources" of PAH releases is indeed a monumental task because of the omipresent nature of petroleum- and coal-derived oils, fuels or solvents that contain at least small amounts of PAHs. The major sources discussed here are summarized in two tables: Coal Tar Production and Distillation (Table 5-8) and Petroleum Sources (Table 5-9); supporting data and related information are presented in Tables 5-10 and 5-11 and Appendix A.

It is important to comment on the quality of available data in this section. Specifically, concentrations of the various PAHs in crude oils, coal, or coal and petroleum products are highly variable, depending upon the geographic source and method of processing. Furthermore, as subsequent calculations are based on these concentrations, they can be considered as estimates only, at the order of magnitude level of reliability.

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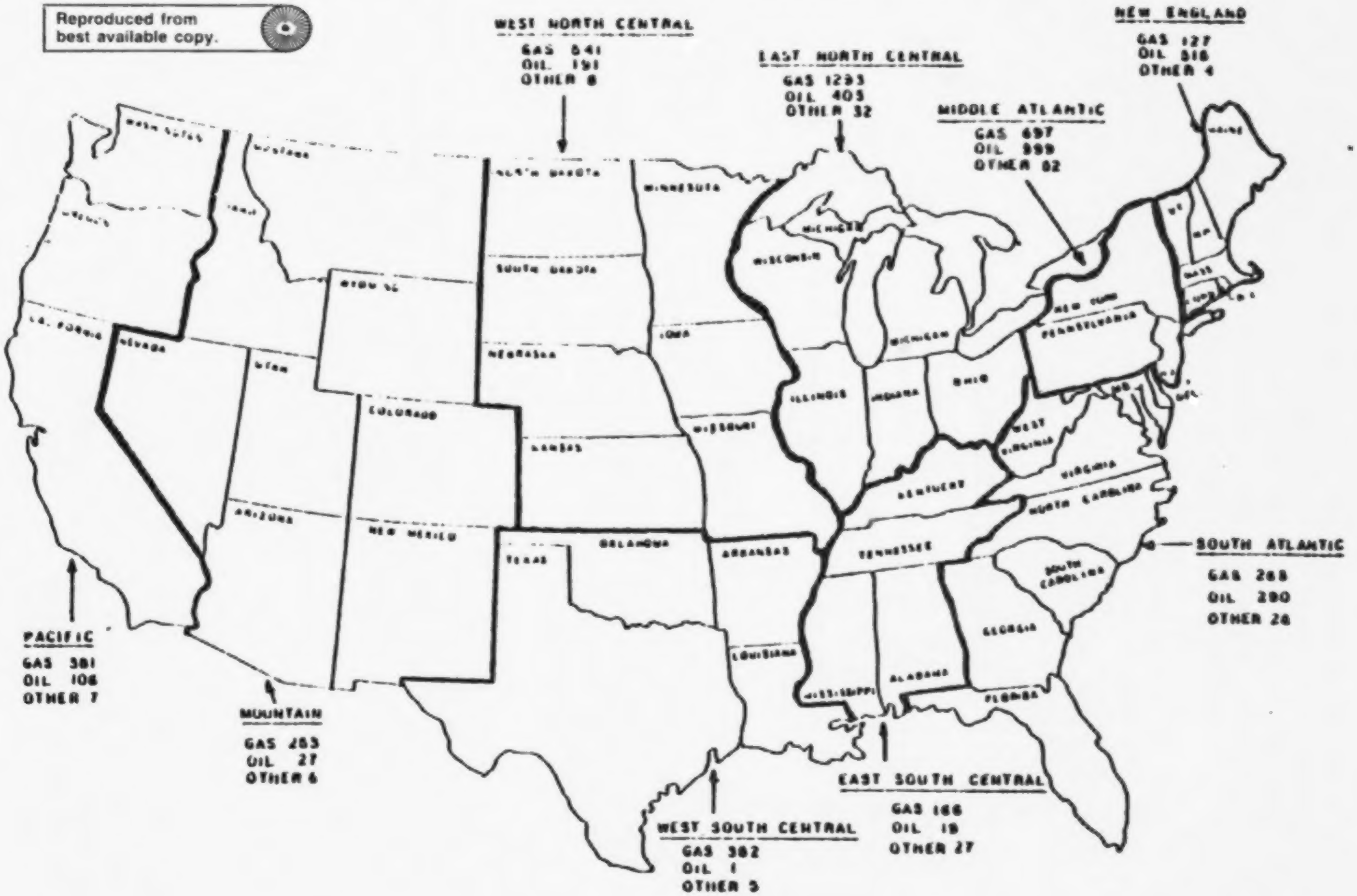

WEST NORTH CENTRAL
GAS 541
OIL 191
OTHER 8
EAST NORTH CENTRAL
GAS 1233
OIL 405
OTHER 32
MIDDLE ATLANTIC
GAS 697
OIL 999
OTHER 82
NEW ENGLAND
GAS 127
OIL 316
OTHER 4
SOUTH ATLANTIC
GAS 268
OIL 290
OTHER 26
EAST SOUTH CENTRAL
GAS 166
OIL 19
OTHER 27
WEST SOUTH CENTRAL
GAS 382
OIL 1
OTHER 5
MOUNTAIN
GAS 283
OIL 27
OTHER 6
PACIFIC
GAS 381
OIL 106
OTHER 7
WASHINGTON
OREGON
CALIFORNIA
NEVADA
IDAHO
MONTANA
WYOMING
UTAH
ARIZONA
COLORADO
NEW MEXICO
NORTH DAKOTA
SOUTH DAKOTA
NEBRASKA
KANSAS
OKLAHOMA
TEXAS
MINNESOTA
IOWA
MISSOURI
ARKANSAS
LOUISIANA
WISCONSIN
MICHIGAN
ILLINOIS
INDIANA
OHIO
KENTUCKY
TENNESSEE
MISSISSIPPI
ALABAMA
GEORGIA
FLORIDA
SOUTH CAROLINA
NORTH CAROLINA
VIRGINIA
WEST VIRGINIA
PENNSYLVANIA
NEW YORK
MAINE
VT
NH
MASS
R.I.
MD
DEL

Figure 5-3. Geographical Distribution of Residential Fuel Consumption for Space Heating, 1975, 10^{15} J/yr (EPA 1979e)

Table 5-8. PAH Materials Balance: Coal Tar Production and Distillation, 1978 (kkg/yr)[a]

		Used By Producers[b]			Sold for Refining[b]				Environmental Releases				
											Water[e]		
	Tar Production[b]	Refining/ Topping	fuel	Others	Quantity	On Hand Dec. 31	Coal-Tar Pitch[c]	Coal-Tar Creosote Oil[d]	Air[f]	Land	POTW	Surface	Total
Acenaphthylene	(no data available)								9	10	10	10	40
Benzo(a)anthracene	neg[g]						8	1	1	2	2	2	7
Benzoperylenes	(no data available)								0.4				neg
Benzo(a)pyrene	77	18	13	1.2	44	6.6	8	neg[g]	2	0.6	0.7	0.9	4
Chrysene	10,000	2400	1700	150	5700	860	8	0.4	3	2	2	3	10
Indenopyrenes	(no data available)								0.4				neg
Total							Total		16	15	15	16	60

a) All production values rounded to two significant figures, environmental releases to one figure; blank spaces = data not available. Totals may not add due to rounding.

b) See Tables A-8, 9 for tar production/distribution totals and concentrations of PAHs, respectively. Density of Coal tar = 1.223 kg/l.

c) See Table A-9 for PAH concentrations; total based on 790 x 10^3 kkg pitch produced in 1978 (USITC 1979).

d) See Table A-9 for PAH concentrations; total based on 3.3 x 10^8 l creosote oil produced in 1978 (USITC 1979). Density of creosote oil = 1.06 kg/l.

e) See Table A-10 for coke plant effluent discharge factors. Distribution of discharge: 33% - direct, 25% - POTWs, 2% deep well, 40% quenching (20% - land, 20% - air) (EPA 1979d). All values rounded to one significant figure.

f) Includes emissions from coke-oven doors (see Table 5-10).

g) Less than 1 kkg.

Table 5-9. PAHs in Contained Petroleum Sources, (kkg/yr)[a]

PAH	Input[b]			Environmental Releases[c]						
				Oil Spills		Gasoline Spills		Petroleum Refinery Wastes		
	Crude Oil[d]	Gasoline[e]	Diesel Fuel[f]	Water	Land	Water	Land	Air[h]	Water	Land
Acenaphthylene	340,000			10	neg[g]					
Benzo[a]anthracene	trace	250	3	neg	neg	neg	neg			
Benzo[b]fluoranthene	<4,200			neg	neg					
Benzo[k]fluoranthene	<4,200			neg	neg					
Benzo[ghi]perylene	17	170	0.9	neg	neg	neg	neg	5		
Benzo[a]pyrene	840	170	2	neg	neg	neg	neg	3		neg
Chrysene	<84,000	170	20	3	neg	neg	neg			
Dibenzo[a,h]anthracene					neg					
Ideno[1,2,3-cd]pyrene					neg					

a) Blanks indicate data not available.
b) See Table A-11 for PAH concentrations.
c) See Appendix A, Note 4, for derivations
d) Based on 8.4 x 10^8 kkg of crude oil consumed (Guerin 1978).
e) Based on 7.4 x 10^6 bbl/day consumed (Oil and Gas Journal 1979); 42 gal/bbl; 0.73 kg/l.
f) Based on 3.4 x 10^{10} l/yr; 0.865 kg/l.
g) Less than one kkg.
h) Based on data in Table A-13, 4.985 x 10^6 bbl/day feed for catalytic cracking, 0.887 x 10^6 bbl/day for catalytic hydrocracking (Oil and Gas Journal 1979), 42 gal/bbl.

Table 5-10. PAH Emissions: Coke-Oven Doors

	Emission Rate (mg/hr/oven)[a]		Yearly Emission (kkg/yr)[b]
Chrysene	16		1.0
Indenopyrene	6		0.4
Benzoperylene	6		0.4
Benzopyrene	16		1.0
		Total	3.0

a) EPA 1977e

b) Based on 1300 kkg coke produced per typical coke oven battery of 58 ovens; 160,000 kkg/day typical capacity for total by-product coke-making industry (resulting in a total of 123 batteries); 365 day, 24 hour operation; emission data in first column, EPA 1979f.

Table 5-11. Water Discharges of Benzo[a]pyrene Group PAHs: Timber Products, 1978 (kkg/yr)[a]

PAH	Input	Raw Discharge		Treated Discharge	
		Concentration (mg/ℓ)	Quantity[b]	Concentration (mg/ℓ)	Quantity[b]
Acenaphthylene	6,900	0.68	5	0.05	neg
Benzo[a]anthracene	neg	0.85	6	0.43	3
Benzo[b]fluoranthene	neg	0.39	3	0.31	2
Benzo[k]fluoranthene	neg	0.45	3	0.03	neg
Benzo[ghi]perylene	neg	0.05	neg	0.01	neg
Benzo[a]pyrene	neg	0.36	2	0.04	neg
Chrysene	0.4	0.69	5	0.90	neg
Dibenzo[a,h]anthracene	neg	0.007	neg	ND	neg
Indeno[1,2,3-cd]pyrene	neg	0.18	1	0.02	neg

a) See Table 5-8, coal-tar creosote.

b) Based on 56% of 476 total plants using creosote or mixture thereof, 350 day/yr, 75,500 liters/day/plant, assumed to go to POTW.

Source: EPA, 1979d.

5.1.3.1 Coal Tar

Coal tar is the heavy distillate fraction from the destructive distillation (coking) of coal. The distribution of coke-oven tar production in the U.S., PAH concentrations, and environmental releases (Tables 5-10. A-8, and A-9) have been combined to provide the information presented in Table 5-8. Naphthalene (see Section 3.1), present in the largest concentration in coal tar, is the only PAH compound warranting recovery and isolation in large amounts. Anthracene is also isolated, but to a lesser extent and usually in crystals which form during creosote oil recovery (see Section 4.1). Creosote oil is used in the wood preserving industry. Information on PAH concentrations in creosote oil and wastewater discharges during use are presented in the previously mentioned tables and Table 5-11, respectively. Of a total of 224 wood preserving plants responding to EPA's data collection protocol, two reported direct discharge, 47 reported discharge to POTWs and the remainder reported self-contained no-discharge operations (mostly evaporation with some oil irrigation or treated effluent recycle; EPA 1979g).

Besides creosote, other principal tar products containing PAHs are pitch and refined tar, used in a variety of applications ranging from road materials and electrodes to shampoos. Coal tar and tar products are also used as fuel (see Table 5-8), either by producers (e.g., iron and steel plants) or distillers.

5.1.3.2 Petroleum Sources

The other fossil fuel source containing appreciable amounts of PAHs is petroleum. The concentration and emissions data in Appendix Tables A-11, A-12 and A-13 have been combined for use in the summary Table 5-9.

Only spills and petroleum refinery environmental releases are presented here; gasoline combustion has been covered in Section 5.2.1 with other combustion sources. As mentioned previously, the type of crude feedstock determines its chemical composition and, therefore, the composition of specific waste streams. Other variables include pollution controls, age of the processes used (level of technology), and operational practices and control. It has been estimated that a total of 0.1 kkg benzo[a]pyrene is released in solid waste streams industry-wide from petroleum refining (EPA 1976). The air emissions also listed in Table 5-9 are specifically from petroleum catalytic cracking, which accounts for over 50% of the annual oil feed to refineries (Oil and Gas Journal 1979); the remainder is used for catalytic reforming, for which emissions factors were not available.

5.1.4 Publicly-Owned Treatment Works

Input of PAHs to POTWs is largely dependent upon variations in industrial discharges feeding the POTWs and the types of industry in a particular municipality. A recent EPA study of 20 urban POTW facilities with secondary treatment and varying feed conditions produced a materials balance of PAHs shown in Table 5-12.

Table 5-12. Materials Balance of Benzo[a]pyrene Group PAHs: Municipal POTWs (kkg/yr)[a]

PAH	Input[b]	Environmental Releases		
		Air[c]	Water[d]	Land[e]
Chrysene	7.3	NA[f]	3.7	9.1
Indeno[1,2,3-c,d]pyrene	3.7	3.7	ND[g]	neg[h]
Benzo[a]pyrene	3.7	3.7	ND	neg
Benzo[b]fluoranthene	3.7	0.7	ND	3
Benzo[k]fluoranthene	3.7	3.7	ND	neg
Benzo[ghi]perylene	11	11	ND	neg
Dibenzo[a,h]anthracene	3.7	3.7	ND	neg
Benzo[a]anthracene	7.3	NA	3.7	9.1
Acenaphthylene	ND	NA	3.7	neg

a) All values rounded to two significant figures.
b) Based on influent concentrations shown in Table 5-13, 10^{11} liters per day total POTW flow.
c) Difference between input and water and land.
d) Based on secondary effluent concentrations shown in Table 5-13, 10^{11} l/day total POTW flow.
e) Based on wet sludge concentrations shown in Table 5-13, 6×10^{6} metric tons dry sludge generated/yr, wet sludge 95% water (by weight).
f) Not available.
g) Not detected.
h) <1 kkg.

Table. 5-13. PAH Concentrations in Municipal POTWs (μg/l)

PAH	Influent	2° Effluent	Raw Sludge
Acenaphthylene	ND	0.1	3
Benzo[a]anthracene	0.2	0.1	76
Benzo[b]fluoranthene	0.1	ND	25
Benzo[k]fluoranthene	0.1	ND	2.5
Benzo[ghi]perylene	0.3	ND	0.6
Benzo[a]pyrene	0.1	ND	3.9
Chrysene	0.2	0.1	76
Dibenzo[a,h]anthracene	0.1	ND	0.2
Indeno[1,2,3-c,d]pyrene	0.1	ND	0.2

a) Average values.

Source: EPA 1980d

The materials balance in Table 5-12 was constructed using a total POTW flow of approximately 10^{11} l/day (EPA 1978c) and the average concentrations of the various PAHs in influent, effluent and sludge, presented in Table 5-13. For purposes of these calculations, influent and effluent flow rates are assumed to be equal, i.e., water losses from sludge removal and evaporation are small compared with influent flows. With these assumptions, an estimated 70 kkg of PAHs were discharged to water from POTWs in 1978, while there was an input of 590 kkg in influent. Of aquatic releases from POTWs, 11 kkg are attributed to BaP group PAHs.

PAHs discharged in sludge can be estimated from the PAH concentrations in sludge and the quantity of dry sludge produced annually: 6.0×10^6 kkg (EPA 1979i). Wet sludge is assumed to be 95% water by weight. As ocean dumping of sludge is mandated to cease by 1981, and if one assumes that more stringent air quality standards curb incinerator use (EPA 1979j), the 25 kkg of the BaP group of PAHs contained in sludge are assumed to be discharged to land.

PAHs released to the atmosphere were estimated by the difference from the above calculations (influent loading - effluent and sludge loading). On this basis, an estimated 30 kkg of these PAHs were released to the atmosphere from POTWs in 1978.

5.1.5 Summary

As shown in Table 5-14, the largest amount, 96% (2700 kkg) of the total releases of BaP group PAHs (2800 kkg), is emitted to the atmosphere. Of the atmospheric emissions, combustion (Table 5-1) is the source of 99% of the BaP group PAHs released. Of the various compounds, releases of acenaphthylene are much greater than the other PAHs in this group. Aquatic discharges that have been identified (37 kkg total) are chiefly the result of oil/gas spills and POTWs (Tables 5-9 and 5-12, respectively). Releases to land (50 kkg) are again mainly due to coal tar production and POTWs.

Table 5-14. Estimated Environmental Releases of BaP Group PAHs (kkg/yr)

	Air	Land	Water		Total
			Surface	POTW	
Acenaphthylene					
combustion[a]	930	–[c]			930
crankcase oil disposal[b]					
coal tar production[d]	9	10	10	10	40
contained petroleum sources[e]		neg	10		10
timber products[f]				neg	
tire wear					
POTW		neg	4		4
Total:	940	10	20	10	980
Benzo[a]anthracene					
combustion	340				340
crankcase oil disposal		1		1	2
coal tar production	1	2	2	2	7
contained petroleum sources		neg	neg		neg
timber products				3	3
tire wear					
POTW		9	4		13
Total:	340	10	6	6	360
Benzo[b]fluoranthene					
combustion	210				210
crankcase oil disposal					
coal tar production					
contained petroleum sources		neg	neg		
timber products				2	2
tire wear					
POTW	1	3	neg		4
Total:	210	3	neg	2	220

a) See Table 5-1.
b) See Table 5-7.
c) Blanks mean data not available; neg means <1.
d) See Table 5-8.
e) See Table 5-9.
f) See Table 5-11.
g) See Table A-4, air releases included in combustion.
h) See Table 5-12.

Table 5-14. Estimated Environmental Releases of BaP Group PAHs (kkg/yr) (Continued)

	Air	Land	Water		Total
			Surface	POTW	
Benzo[k]fluoranthene					
combustion[a]	210				210
crankcase oil disposal[b]		1.5	-	1.5	3
coal tar production[d]					
contained petroleum sources[e]		neg	neg		neg
timber products[f]				neg	neg
tire wear					
POTW	4	neg	neg		4
Total:	210	2	neg	2	220
Benzo[ghi]perylene					
combustion	310				310
crankcase oil disposal		1.5		1.5	3
coal tar production	neg				neg
contained petroleum sources	5	neg	neg		5
timber products				neg	neg
tire wear	8	7			15
POTW	11	neg	neg		11
Total:	330	9	neg	2	340
Benzo[a]pyrene					
combustion	160				160
crankcase oil disposal		neg		neg	neg
coal tar production	2	0.6	0.9	0.7	4
contained petroleum sources	3	neg	neg		3
timber products				neg	neg
tire wear	1	1			2
POTW	4	neg	neg		4
Total:	170	2	1	1	170

Table 5-14. Estimated Environmental Releases of BaP Group PAHs (kkg/yr) (Continued)

	Air	Land	Water Surface	Water POTW	Total
Chrysene					
combustion[a]	340				340
crankcase oil disposal[b]		1		1	2
coal tar production[d]	3	2	3	2	10
contained petroleum sources[e]		neg	3		3
timber products[f]				neg	neg
tire wear					
POTW		9	4		13
Total:	340	10	10	3	370
Dibenzo[a,h]anthracene					
combustion	40				40
crankcase oil disposal					
coal tar production					
contained petroleum sources		neg			neg
timber products				neg	neg
tire wear					
POTW	4	neg	neg		4
Total:	40	neg	neg	neg	40
Indeno[1,2,3-c,d]pyrene					
combustion	130				130
crankcase oil disposal					
coal tar production	neg				neg
contained petroleum sources		neg			neg
timber products				neg	neg
tire wear	1	2			2
POTW	4	neg	neg		
Total:	130	1	neg	neg	130

5.2 FATE AND DISTRIBUTION IN THE ENVIRONMENT

5.2.1 Introduction

This section describes the fate processes that determine the ultimate distribution of BaP and the other related PAHs in the aquatic environment and, therefore, the opportunities for water-borne exposure to humans and other biota. Much of the information presented pertains specifically to BaP; however, since the properties and fate characteristics of this compound are similar to those of the other compounds in the group, the behavior of BaP in the environment is believed to be a good model. In addition, some information concerning benz[a]anthracene is presented from a major study of the fate of organics in the environment (Smith et al. 1978).

Section 5.2.2 presents an overview of the environmental loading of BaP to the aquatic media, both direct releases to surface water and deposition from the atmosphere. In Section 5.2.3, physical/chemical properties of the BaP group PAHs are summarized in order to identify the processes that transform and transport the chemical upon its release to the environment (Section 5.2.3.1). Section 5.2.3.2 discusses the interplay of fate processes that determines the major pathways of BaP in aquatic environmental media.

Modelling efforts were undertaken based upon environmental loadings estimated in Section 5.1, in order to characterize the fate and distribution of BaP in specific environmental scenarios; these are discussed in Section 5.2.3.3. Monitoring data from STORET and a limited number of other surveys are summarized in Section 5.2.4 to provide indications of concentrations of BaP group PAHs actually detected in aquatic media. Finally, Section 5.2.5 summarizes those aspects of the fate and ultimate environmental distribution of BaP having the greatest significance for the water-born exposure of humans and other biota.

5.2.2 Inputs to Aquatic Media

5.2.2.1 Atmospheric Deposition

Data presented in Section 5.1 indicate that direct releases of BaP to surface waters are negligible; atmospheric emissions of BaP from combustion sources were estimated to be approximately 170 kkg in 1978 (Section 5.1). Atmospheric BaP may be transported to the aquatic environment indirectly via wet and/or dry deposition. These physical removal mechanisms are expected to be significant for BaP; Cupitt (1980) suggests an atmospheric residence time (time required for BaP to be reduced to 1/e of its original value) of approximately 8 days.

The air-to-surface transport pathway has been evaluated for BaP and is detailed in Appendix B; the results of that analysis are summarized in Table 5-15. Under ambient conditions in either rural or urban areas, virtually all of the BaP is adsorbed onto aerosols. The deposition

TABLE 5-15. EVALUATION OF AIR-TO-SURFACE PATHWAY FOR BENZO[a]PYRENE

	Rural	Urban
Adsorbed fraction of airborne mass	0.99	1
Dry deposition Velocity (cm/sec)	1	1
Precipitation scavenging ratio: $\left(\frac{\text{ng/l (water)}}{\mu\text{g/m}^3\text{ (air)}}\right)$	$6\text{x}10^4$	$6\text{x}10^4$-$1.2\text{x}10^5$ [a]
Percent of atmospheric emissions deposited		
- dry deposition	22	19
- wet deposition	4	4-7
- total	26	23-26

[a] Depends on distance from combustion source.

parameters for BaP vary only slightly from rural to urban environments, since they are highly dependent upon vapor/aerosol partitioning. As shown in Table 5-15, approximately 26% of atmospheric BaP will be deposited on the surface. The analysis indicates that dry deposition of BaP adsorbed onto atmospheric aerosols accounts for most of the removal; wet deposition is less significant by a factor of from three to five, depending upon the extent of equilibrium achieved in the combustion plume.

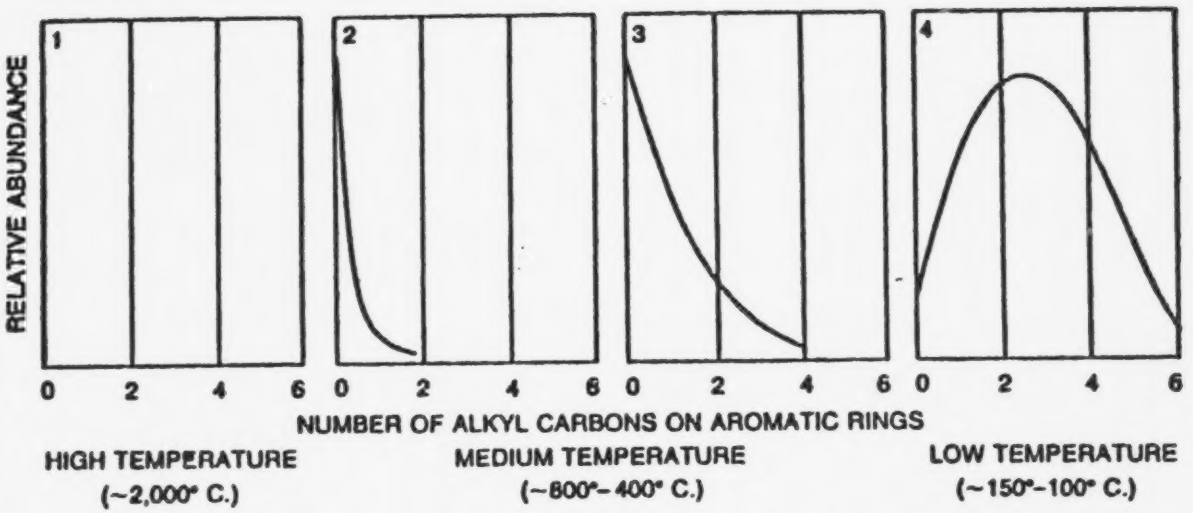
On the basis of total annual atmospheric emissions of 170 kkg for 1978 (Section 5.1), approximately 44 kkg may have been deposited on the surface of the U.S. Some of the fallout will land on surface waters. If approximately 2% of the total area of the continental U.S. is surface water (U.S. Bureau of the Census 1980), direct deposition onto aquatic systems would have amounted to less than 1 kkg.

A fraction of the remaining fallout deposited on land would be transported ultimately to aquatic systems via surface runoff. Data cited by Neff (1979) suggest that 500 kkg/yr of BaP are deposited on surface waters worldwide (including oceans) and that 118 kkg/yr end up in aquatic systems via surface runoff. These ratios indicate that surface runoff would deposit much less than 1 kkg of BaP in U.S. waters each year. The local effects of surface runoff, especially near combustion sources, could still be significant. The BaP adsorbed to surface particles is tightly bound and would probably be carried along with these particles in runoff. However, there are insufficient data to estimate the importance of this pathway.

5.2.2.2 Source Identification by PAH Composition

Sediments have an integrating effect on patterns of PAH input over time and supply geographical information when current patterns, sediment origins, and settling rates are known (Dunn and Stich 1976). It should, however, be mentioned that the composition in sediment does not always directly reflect the original PAH composition at the source. Partitioning between the sediment and aqueous phases may be different for parent PAHs and alkylated PAHs within an aromatic series, since solubility in water decreases as the number of alkyl carbons increases (Armstrong *et al.* 1977).

Information on whether the origin of PAHs in water is atmospheric deposition from combustion sources or direct discharge can be gained from studying the PAH composition in the sediments. The extent of alkylation of PAH mixtures has been shown to be highly dependent upon the combustion temperatures (Blumer 1976). The number of alkyl carbons on PAHs formed during combustion of fuel (400°C-2000°C) is generally 0-2; at the low temperatures at which petroleum was formed over geologic time (100°C-150°C), PAHs with 2-4 alkyl carbons are most common (Figure 5-4).



Source: Blumer (1976)

FIGURE 5–4 EFFECT OF COMBUSTION TEMPERATURE ON RELATIVE ABUNDANCE OF ALKYL CARBON ATOMS ON PRODUCED POLYNUCLEAR AROMATIC HYDROCARBONS

Furthermore, it has been reported that the distribution of homologs in PAHs measured in soils and marine sediments varies little over a wide range of depositional environments. Homologs extending to at least seven alkyl carbons occurred in all samples and all PAH series. However, within a PAH series, the unsubstituted hydrocarbons were the most abundant with the intensities of isomeric homologs, decreasing nearly two-fold with each additional carbon atom (Blumer and Youngblood 1975). In one study of PAH composition of sediment, a semilog plot of abundance vs. number of alkyl carbons was found to produce a straight line with a highly negative slope (Hase and Hites 1977). These data are presented in Figure 5-5, along with similar measurements for river water and air particulates. The authors postulated that the steep slope characteristic of urban air particulates was changed to the more gradual slope of the sediments due to the differential solubilities and partitionings of parent PAHs and their alkyl homologs. After airborne particulates are deposited on soil or water, the lower homologs continuously fractionate into the water phase, thereby increasing the relative abundance of the alkylated species in the sediment. The slope of the airborne particulate plot is intermediate between those of water and sediment, and this led the authors to the conclusion that the main source of the aquatic load of PAHs is anthropogenic, airborne PAHs produced during combustion.

It should be noted that industrial effluents containing PAHs would undergo similar fractionation (Lewis 1975, Armstrong et al. 1977). PAH profiles from petroleum sources show that parent compounds of each series are less abundant than the alkylated forms. The fact that the unsubstituted PAHs are the most abundant homolog (despite higher aqueous solubility) in sediment from non-industrial areas further indicates that the major source of PAHs in sediment is generally a combustion source rather than direct contamination by fossil fuels. Sediment known to be contaminated with fossil fuels exhibited an elevated concentration of four- and five-ring PAHs (due to lower water solubility) similar to that in the uncontaminated sediments, but also contained a much higher proportion of alkylated compounds (Youngblood and Blumer 1975).

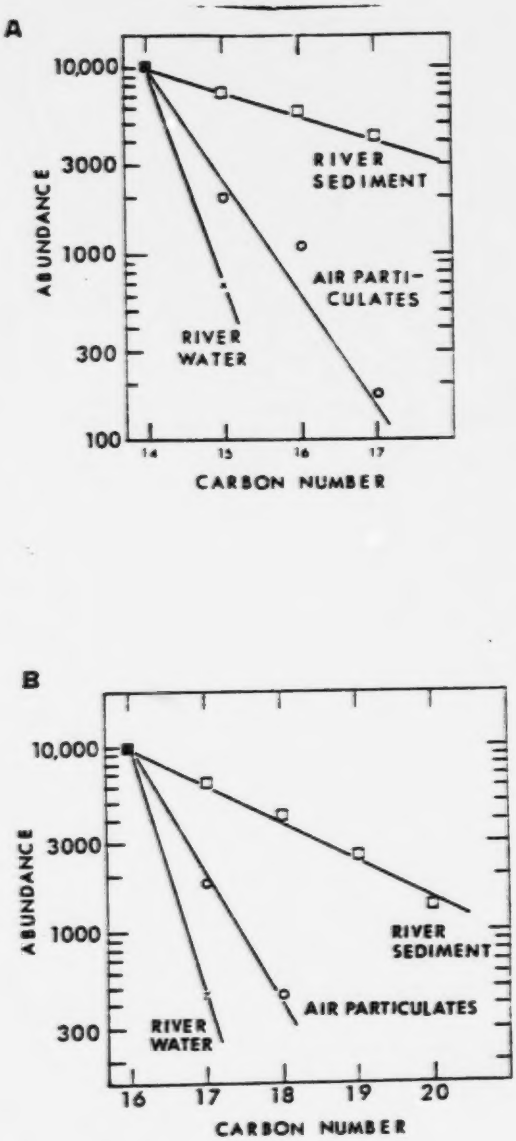
5.2.3 Environmental Fate

5.2.3.1 Basic Physical/chemical Properties

The physical/chemical properties that are relevant to the fate and distribution of the BaP group PAHs are summarized in Table 5-16. The properties of all of these PAHs, exclusive of acenaphthylene, are quite similar; the properties of acenaphthylene more closely resemble the compounds discussed in Chapters 3.0 and 4.0 of this report.

5.2.3.2 Pathways in the Aquatic Environment

This section examines the details of the fate pathways of BaP in the aquatic environment. The major processes for removal from the water



Source: Hase and Hites (1977).

Note: Data are normalized such that the unsubstituted species is 10,000 units in each case; A-pyrene, B-phenanthrene.

FIGURE 5–5 DISTRIBUTION OF ALKYL HOMOLOGS IN THE PHENANTHRENE-TYPE SERIES AND PYRENE-TYPE SERIES IN RIVER WATER, AND AIR — CHARLES RIVER, BOSTON, MA

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TABLE 5-16. BASIC PHYSICAL/CHEMICAL PROPERTIES OF BENZO[a]PYRENE GROUP PAHS

	Benzo[a]pyrene	Acenaphthylene	Benz[a]-anthracene	Benzo[b]-fluoranthene	Benzo[k]-fluoranthene	Benzo[g,h,i]-perylene	Chrysene	Dibenz[a,h]-anthracene	Indeno[1,2,3-c,d]-pyrene
Abbreviation	BaP	-	BaA	BbF	BkF	Bg,h,iPr	Chr	dBahA	I1,2,3-c,dP
Formula	$C_{20}H_{12}$	$C_{12}H_{8}$	$C_{18}H_{12}$	$C_{20}H_{12}$	$C_{20}H_{12}$	$C_{22}H_{12}$	$C_{18}H_{12}$		$C_{22}H_{12}$
MW	252.32^{c}	152.21^{c}	228.28^{c}	252.32^{c}	252.32^{c}	276.34^{c}	228.28^{c}	278.36^{c}	276.34^{c}
Melting Pt. (°C)	179^{b}	92^{b}	155-157^{b}	167-168^{b}	217^{b}	222^{b}	256^{b}	270^{b}	162.5-164^{b}
Boiling Pt. (°C)									
Vapor Pressure (torr)	$5.6\times10^{-9}\pm(25°C)^{a}$ $5\times10^{-9}(20°C)^{b}$	$0.030(25°C)^{a}$ $10^{-3}-10^{2}(20°C)^{b}$	$2.2\times10^{-8}(25°C)^{a}$ $5\times10^{-9}(20°C)^{b}$	$5\times10^{-7}(25°C)^{a}$ $10^{-11}-10^{-6}(20°C)^{b}$	$5\times10^{-7}(25°C)^{a}$ $9.6\times10^{-11}(20°C)^{b}$	$1\times10^{-10}(25°C)^{a}$	$6.3\times10^{-9}(25°C)^{a}$ $10^{-11}-10^{-6}(20°C)^{b}$	$1.0\times10^{-10}(25°C)^{a}$	$1\times10^{-10}(25°C)^{a}$
Water Solubility mg/l at 25°C	0.0038^{a}	3.93^{b}	0.0057(20°)^{a} 0.014^{b} 0.009^{b}	0.001^{a}	0.00055^{a}	0.0003^{a} 0.00026^{b}	0.0018^{a} 0.002^{b}	0.0005^{b}	0.0005^{a}
Log K_{ow}, 25°C	6.08^{a} 6.04^{b}	3.72^{a} 4.07^{b}	5.61^{a,b}	6.08^{a} 6.57^{b}	6.08^{a} 6.84^{b}	6.51^{a} 7.21^{b}	5.61^{a,b}	6.84^{a} 5.97^{b}	6.51^{a} 7.66^{b}
Log K_{oc}, 25°C		3.46^{a}	5.34^{a}				5.34^{a}		
Henry's law constant at 25°C (atm m^{3}/mole)	4.89×10^{-7}	1.52×10^{-3}	7.34×10^{-7}	1.66×10^{-4}	3.02×10^{-4}	1.21×10^{-7}	1.05×10^{-6}	7.3×10^{-8}	6×10^{-10}

[a] SRI (1980).
[b] Callahan et al. (1979)

[c] Weast (1974).

column are reviewed first, i.e., volatilization and sedimentation; the transformation and degradation pathways for BaP in solution are then described. Finally, biodegradation and its role in determining the ultimate fate of PAHs, especially in sediment, are considered.

Volatilization

Benzo[a]pyrene has a vapor pressure of 5×10^{-9} torr at 25°C and a Henry's law constant of $4.8 \times 10^{-7} \frac{m^3 \cdot atm}{mol}$, and these properties suggest that volatilization will not be an important pathway for this compound in the environment. When the role of volatilization in removing BaP and benzo[a]anthracene from the aquatic environment was studied, these two high-molecular-weight PAHs were not rapidly volatilized. With maximum wind (4 m/sec) and current velocity (1 m/sec), half-lives due to volatilization were 150 hours for benzo[a]anthracene and 430 hours for BaP, compared with 3.2 hours for naphthalene and 16 hours for anthracene (Southworth 1979). The variability of volatilization was also examined; for both of these PAHs, a ten-fold increase in current velocity roughly doubled volatilization, while a ten-fold increase in wind velocity increased volatilization five-fold. These larger PAHs appear to be much less sensitive to changes in current velocity and slightly more sensitive to wind velocity changes when compared with lower-molecular weight PAHs.

In a separate study of the environmental fate of these PAHs, the volatilization half-lives were determined to be 89 hours for benz[a]anthracene and 22 hours for BaP under conditions of rapid stirring (Smith *et al*. 1978). In the same study, volatilization half-lives were calculated using a one-compartment model:

	Volatilization Half-life (hours)			
	River	Eutrophic Pond	Eutrophic Lake	Oligotrophic Lake
benzo[a]pyrene	140	350	700	700
benz[a]anthracene	1000	1000	1000	1000

For these compounds, volatilization is a slow process in comparison with the degradation pathways, such as photolysis, discussed below. Furthermore, most of these larger PAHs will be found sorbed onto sediment, and volatilization of sorbed material is presumed to be very slow (Smith *et al*. 1978). Another study of fate and transport of radiolabeled BaP, conducted in a laboratory model ecosystem (Lu *et al*. 1977) failed to detect any radioactivity in traps; this supports the premise that volatilization is not a significant fate process for BaP.

As shown in Section 5.2.3.1, most of the other PAHs in this group exhibit vapor pressures and Henry's law constants that are fairly close to those for BaP and benzo[a]anthracene and would be expected to act similarly with respect to volatilization. Acenaphthylene, on the other hand, will volatilize more rapidly as indicated by its vapor pressure and Henry's law constant.

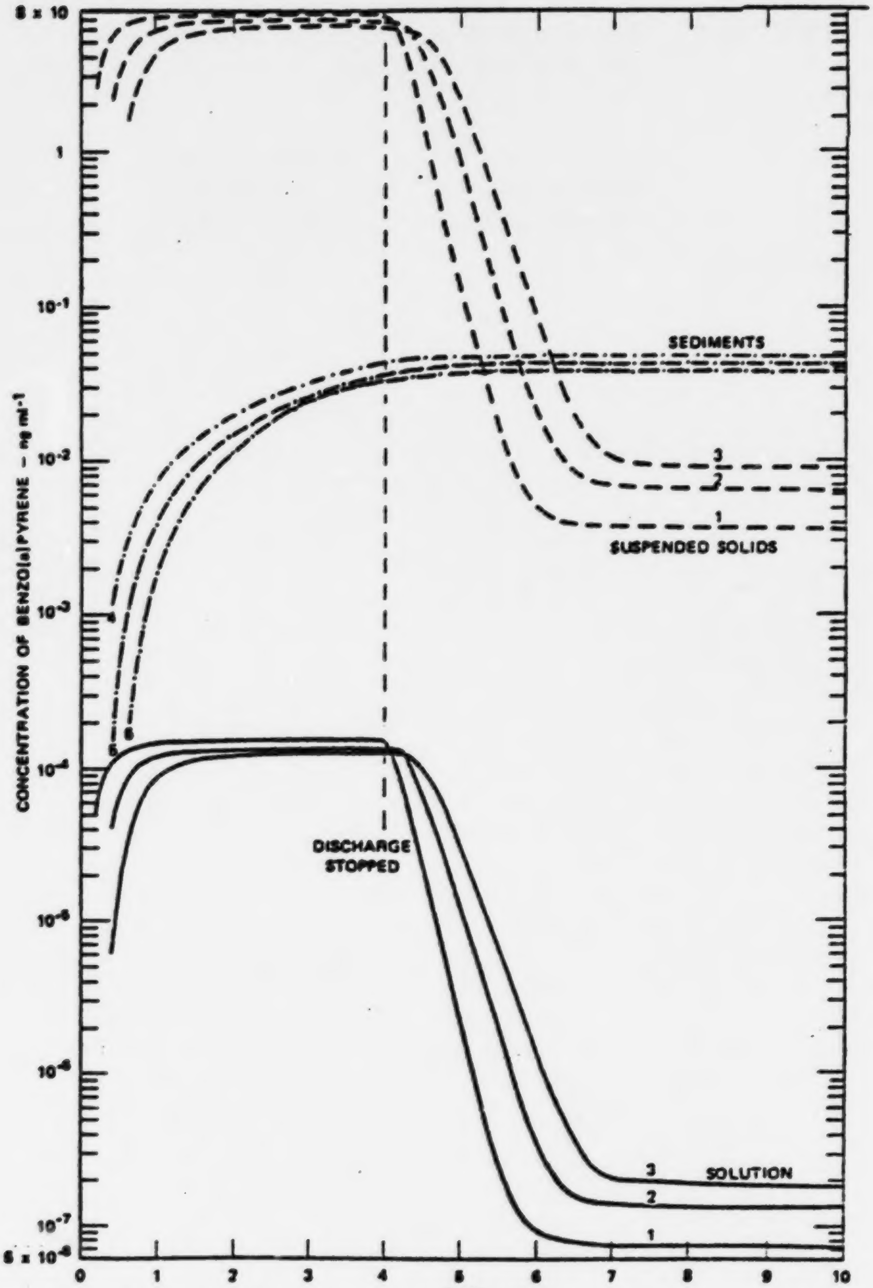
Adsorption, Sedimentation, and Solubilization

Partition coefficient and solubility data suggest that BaP in the aquatic environment is likely to be adsorbed onto sediment and biota. Concentrations (in rivers, ponds and lakes) predicted from a one-compartment model show that the expected concentrations of BaP and benzo[a]anthracene on sediments and suspended solids are greater than the dissolved concentrations by a factor of more than 10^4 (Smith et al. 1978). This is expected since the partition coefficients for all of the PAHs in this group range from 5.61 to 7.66, with the exception of acenaphthylene at 3.72.

The patterns of buildup and decline of BaP in pond, river, and lake simulations were also analyzed by Smith et al. (1978). These predictions are shown in Figure 5-6 for a river system. In all aquatic systems modeled, the concentrations of dissolved BaP rapidly approached steady-state before and after the discharge ceased; sediment concentrations changed very slowly (see Figure 5-6). Cessation of the discharge was shown to cause a 100-1000-fold decrease in dissolved BaP concentrations (solution). Continuous desorption from sediments is sufficient to maintain a low concentration of BaP in the water column, roughly equal to the background level in groundwaters (Suess 1976, Andelman and Snodgrass 1974). The authors obtained similar results in modeling of benzo[a]anthracene (Smith et al. 1978).

BaP sorption onto sediments was shown to be strongly correlated with the organic carbon content of the sediment and not related to the cation exchange capacity (Smith et al. 1978). Since calcium montmorillonite clay (0.06% carbon) displayed some affinity for BaP, it seems likely that BaP sorption involves weak interaction with solid surfaces, as well as strong binding to organic matter. Sorption onto bacterial cells was also investigated, and very high sorption coefficients were obtained. These results are in agreement with other data indicating that PAHs will preferentially sorb onto organic and biological material (Southworth 1977). Sorbent data and partition coefficients for BaP are presented in Table 5-17.

It appears that very little of the BaP in aquatic systems will be found in solution, and this compound can be expected to accumulate in the sediments of placid lakes and reservoirs. River-borne BaP, however, will be transported to the ocean, where inshore and alongshore currents combine to restrict suspended matter to continental areas and spread the PAHs almost uniformly along the coastal regions. In these areas, continuous resuspension and transport via wave action and currents have



Source: Smith *et al.* (1978).

FIGURE 5–6 RESULTS OF MODELING OF THE EFFECT OF SUSPENDED SOLIDS ON THE CONCENTRATION OF BENZO[a]PYRENE IN A PARTIALLY MIXED RIVER SYSTEM

TABLE 5-17. BENZO[a]PYRENE PARTITION COEFFICIENTS FOR VARIOUS SORBENTS

Sorbent	Total Organic Carbon (%)	Carbon Exchange Capacity (meq/100g)	Partition Coefficient Kp
Ca-montmorillonite Clay	0.06	69	1.7×10^4
Des Moines River Sediment	0.6	10.5	3.5×10^4
Coyote Creek Sediment	1.4	13.5	7.6×10^4
Searsville Pond Sediment	3.8	34.5	1.5×10^5
Mixed Bacteria (dry wt. equivalent to 97 mg/l)			$3\text{-}4 \times 10^4$

Source: Smith *et al*. 1978.

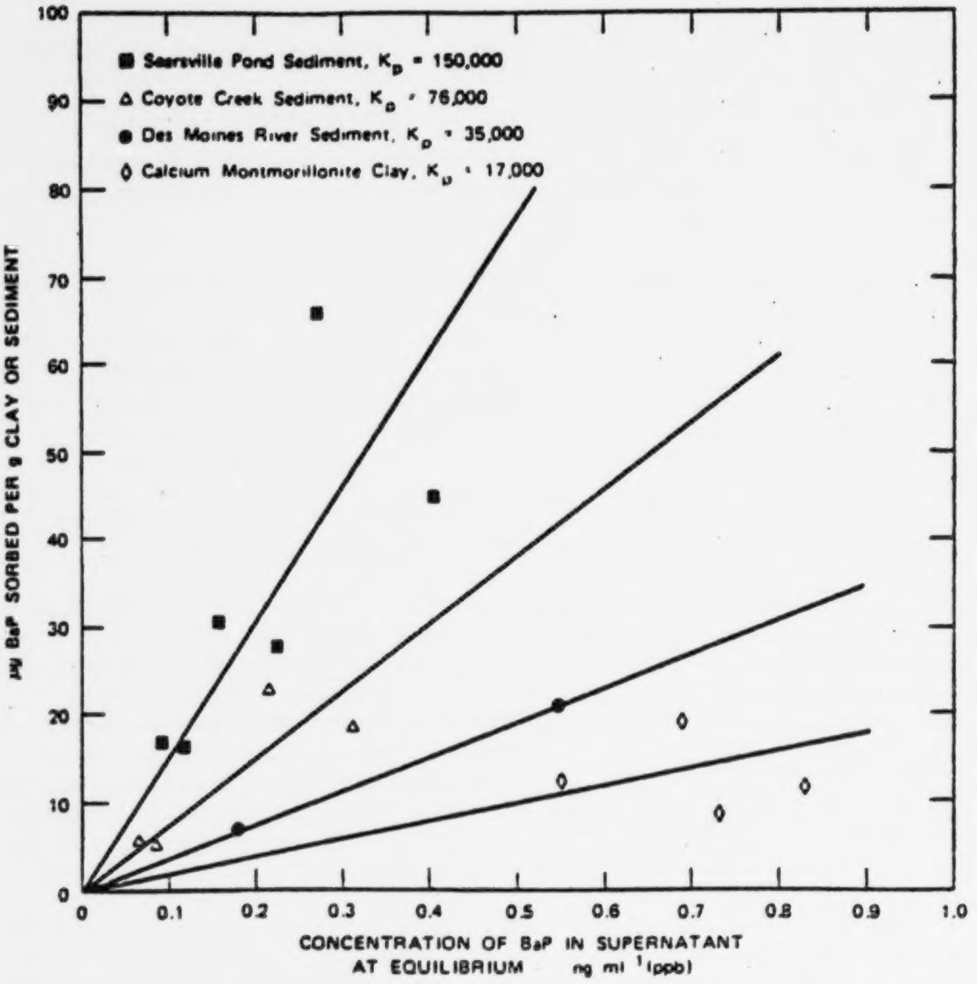
been observed (Gross 1970). PAHs have been identified in coastal waters adjacent to or distant from developed areas (Mallet 1961); coastal plankton have also been found to contain significant amounts of BaP, whereas plankton from the open ocean were uncontaminated (DeLima-Zanghi 1968). Both of these observations indicate that physical transport along the coast is an important fate process for adsorbed PAHs.

The fate of adsorbed PAHs in the water column is influenced by a number of factors; duration of PAH exposure to sunlight will largely determine the extent of photo-oxidation (discussed below). The duration of exposure is partially controlled by particulate sedimentation and resuspension. It has been estimated that estuarine sediment a few millimeters deep is recycled through the water column daily, and that the top 2 cm are recycled annually. Particulates less than 0.5 mm in diameter may reside in the water column for 200-600 years. It has also been postulated that it will take 500-1000 years to bury a single layer of particulate. These data are relevant to determining the fate of adsorbed PAHs since much of this material will ultimately find its way to the coastal regions (Gross 1970).

The low solubility and high K_{ow} for BaP do not necessarily mean that all BaP is sorbed; many natural waters and industrial effluents also contain appreciable amounts of organic material that may increase the solubility of BaP. PAHs could be solubilized by incorporation into micelles if a critical micellar concentration is reached. The concentration of detergents, such as linear alkylbenzene sulfonate, must reach 10-50 mg/l before significant solubilization of BaP occurs; detergent concentrations of 0.1-1.0 mg/l, which are probably closer to environmental concentrations, have no effect on BaP (Bohm-Gasol and Kruger 1965, Il'nitskil and Ershova 1970, Il'nitskil et al. 1971).

Another mechanism for solubilization of PAHs in water exists as a result of the presence of other organic compounds not associated with colloid or micelle formation. With some specific organic mediators, such as butyric and lactic acids, organic solvents, and nitrogen-containing organics, the extent of solubilization has been reported to be proportional to the concentration of the mediator or the square of its concentration (Neff 1979). However, naturally occurring dissolved organic matter (humic acids, fulvic acids, etc.) in seawater appeared to have no effect on solubility of phenanthrene and anthracene (Boehm and Quinn 1973) and may have little effect on PAH solubility, in general.

Sorption of PAHs and other chemicals to natural sediments and biogenous materials has been shown to be a reversible equilibrium process (Smith et al. 1978). Elevated particulate PAH concentrations are generally accompanied by elevated concentrations of dissolved PAHs as shown in Figure 5-7 for BaP. These data do suggest an exchange between adsorbed and dissolved states, and further indicate that there may be a significant amount of BaP in solution, particularly in heavily contaminated systems.



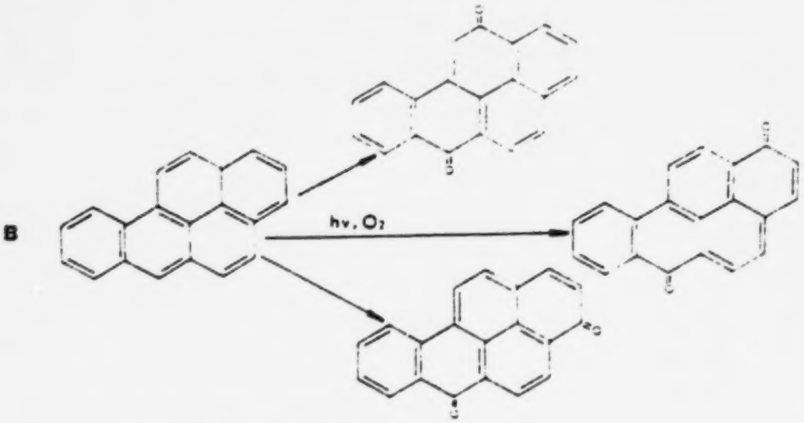
Source: Smith et al. (1978).

FIGURE 5–7 SORPTION ISOTHEMS FOR BENZO[a] PYRENE

The larger-molecular-weight PAHs discussed in this section, particularly BaP, will tend to adsorb onto particulate matter and will ultimately accumulate in the sediment due to sedimentation; half-lives in sediments are expected to be on the order of a few years (White and Vanderslice 1980). Chemical degradation pathways for BaP dissolved in aquatic environments are discussed below; however, these are generally less important as fate processes than sedimentation.

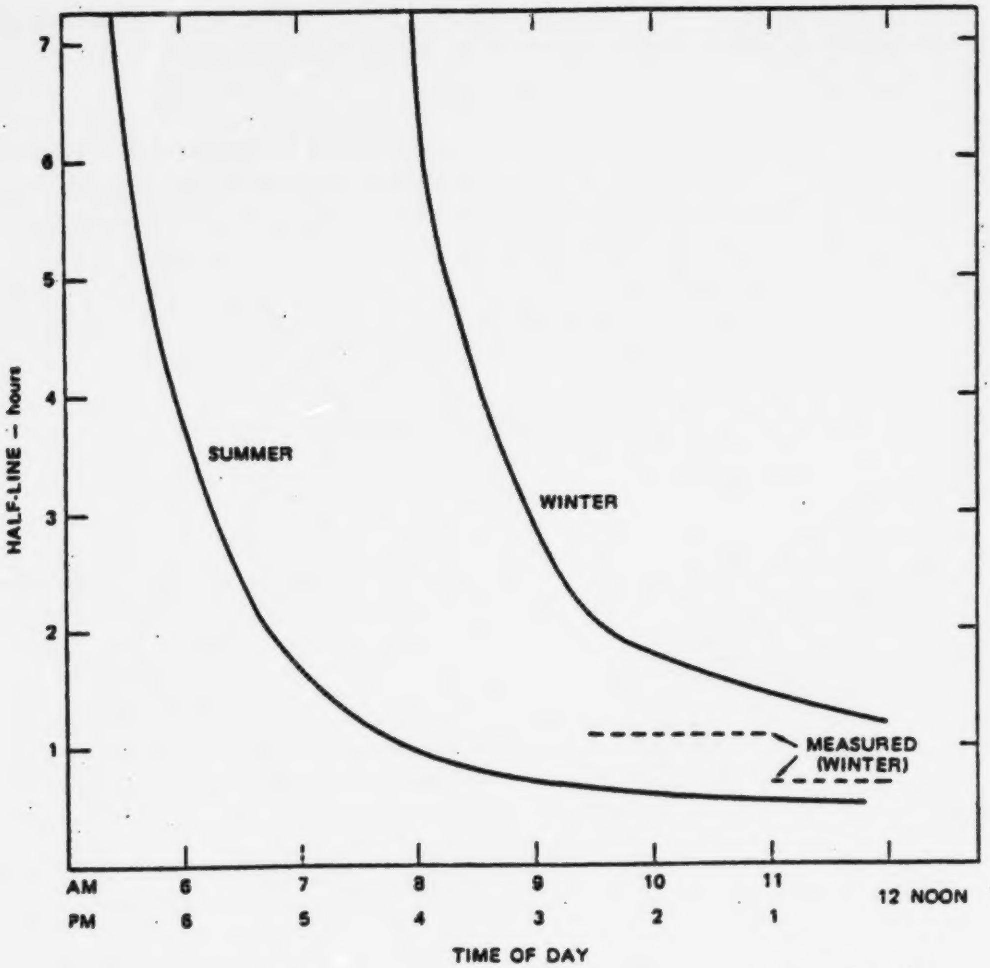
Chemical Degradation

In oxygenated water, photolysis of BaP by light with wavelengths in the solar region gives a mixture of three quinones, as shown below.



Several authors (Andelman and Suess 1970, Smith et al. 1978, NAS 1972, Stevens and Algar 1968, Neff 1979) have suggested that direct photo-oxidation of PAHs is a major oxidation pathway in water. This reaction is postulated to be mediated by singlet oxygen formed by energy transfer from the electronically excited aromatic molecule in its triplet state. However, contrasting evidence has recently been reported (Zepp and Scholtzhauer 1979), suggesting that neither molecular oxygen nor singlet oxygen is the exclusive mediator in any of the major photochemical reactions of the PAHs.

The half-life for direct photolysis of BaP in sunlight, as a function of the time of day was calculated by the procedure of Zepp and Cline (1977) using a quantum yield of 8.9×10^{-4} and the measured UV spectrum of BaP (Smith et al. 1978). Their results are presented in Figure 5-8. (These data were obtained from BaP in a solution of 20% acetonitrile in pure water.) The calculated half-life of 1.2 hours for midday photolysis in winter is in close agreement with the measured results of 1.1 hours and 0.7 hours. Similar experiments with benz[a]-anthracene yielded a half-life of 2 hours for that compound.



Source: Smith et al. (1978).

FIGURE 5–8 CALCULATED SEASONAL AND DAILY VARIATION OF PHOTOLYSIS HALF-LIFE OF BENZO[a] PYRENE

Benzo[a]pyrene in natural waters or pure water containing humic acid exhibited slower rates of photolysis than in similar experiments conducted in pure water (Smith et al. 1978). The rate of photolysis of BaP in the presence of humic acid was five times slower than photolysis in pure water; natural water caused an intermediate, but definite retardation of BaP photolysis. A light-screening effect is probably not entirely responsible since absorbance of natural waters at 366 nm is less than 0.02: the 0.11 absorbance of the humic acid solution should only reduce the photolysis rate by 13%. The authors speculated that substances present in natural waters may quench a BaP excited state, singlet oxygen, or any excited state complex occurring in the reaction. Another possible cause is formation of a complex of BaP with natural organic or inorganic substances in solution, which may alter the reactivity of ground state BaP itself. Evidence for this mechanism was found in that only 50% of the 270 ng/ml of BaP in solution with 30 µg/ml of humic acid could be recovered from aqueous solution by hexane extraction (Smith et al. 1978).

Inhibition of photolysis was also observed when BaP was adsorbed onto Kaolinite clay (McGinnis and Snoeyink 1974); products formed during photolysis are thought to be responsible. Mechanisms by which such effects occur may include competitive reactions of the oxidizing agent(s) formed with the organic matrix. Similar results were obtained in studies of BaP dissolved in acetone and adsorbed onto calcium carbonate (Andelman and Suess 1970).

In another study, BaP adsorbed onto calcite particles in water was exposed to illumination roughly equivalent to one-fourth of the winter solar radiation at the earth's surface (Suess 1972). Half-lives of BaP were 15 hours when the water sample was exposed to atmospheric air, 11 hours under oxygen atmosphere, and 35 hours under helium atmosphere. These results indicate a dependence of BaP degradation on oxygen concentration in water; no oxidation was observed in the absence of sunlight. Again, all of these half-lives are significantly longer than the photolysis half-life of 1-2 hours predicted above.

Benzo[a]pyrene suspended in the water column as a condensed particulate was shown to decompose rapidly under normal daylight conditions (McGinnis and Snoeyink 1974), the rate of decomposition being governed by particle size. For particulates 1.5 mm in diameter, the reaction exhibited first-order kinetics until 55-65% of total BaP was decomposed; at this point, a residual was left that was not affected by an increase in radiant energy. Apparently the decomposition products formed a protective barrier, preventing further reaction of the residual BaP. The authors postulated that in particulate of greater than 0.4-mm diameter a residual would remain. Benzo[a]anthracene did not exhibit this effect; after a threshold of sunlight had been attained, the reaction went to completion, presumably due to the solubility of the decomposition products.

The free radical oxidation of BaP was studied under experimental laboratory conditions at 50°C in order to obtain a first-order rate constant of 5.7×10^{-5} sec^{-1} (Smith et al. 1978). Extrapolating these data to 25°C, this rate corresponds to a second-order rate constant of 1.86×10^{-3} M^{-1} sec^{-1}; using Smith's estimate of 10^{-10} M $RO_2 \bullet$ the half-life was calculated to be 4.3 days ($\sim$100 hours). This half-life is considerably shorter than that estimated by Radding et al. (1976). However, the fact that both are relatively long suggests free radical oxidation of BaP is not competitive with photolysis and adsorption under environmental conditions.

A number of authors have studied the oxidation of PAHs by chlorine (Perry and Harrison 1977) and ozone (Il'nitskii et al. 1968) and the data have been summarized by Radding et al. (1976). The data in Table 5-18 indicate that oxidation by chlorine and ozone may be significant fate processes when these oxidants are available in sufficient quantities, such as in water treatment plants. The products of aqueous chlorination of PAH solutions are not fully known.

Predictions of the fate of BaP in natural waters, based on studies performed under laboratory conditions using pure water or organic solvents, must be made with caution since there may be numerous factors affecting the photo-oxidation rate. The half-lives for individual transformation and removal processes were calculated for various water systems using a one-compartment model (Smith et al., 1978), and are shown in Table 5-19. Chemical degradation due to photolysis is clearly the major transformation pathway for BaP in solution, since the half-lives are at least an order of magnitude smaller than the half-lives for other pathways. Sorption rates were not measured, but were postulated to be at least 100 times faster than photolysis rates; therefore, it is probable that most of the BaP is removed to the sediments.

Biological Fate

Analyzing the exposure and bioavailability of PAHs to aquatic organisms requires an examination of the disposition of these compounds in the biological compartments of the environment. The considerations discussed here include bioaccumulation in aquatic organisms, both as seen in the laboratory and in the field, biotransformation, and biodegradation.

Bioaccumulation

The uptake and bioaccumulation of BaP have been investigated by several authors, using both laboratory model ecosystem studies and predictions based on the octanol:water partition coefficient ($K_{ow} \approx 6$) and water solubility (0.0038 mg/l of BaP. A wide range of bioconcentration data has resulted; some of the data are presented in Table 5-20. As shown, there are significant differences in bio-

TABLE 5-18. RELATIVE HALF-LIVES OF BENZO[a]PYRENE GROUP PAHs IN REACTION WITH MAJOR OXIDANTS

Compound	Half-life (hours) in Reaction with					
	$RO_2\cdot$ 10^{-10} M	Singlet Oxygen	Ozone (water) 10^{-4} M	Ozone (air) 2×10^{-9} M	Cl_2	$HO\cdot$
Benzo[a]pyrene	2.4×10^5	5	1.05	870	$t_{1/2} \sim 0.5$hr for all PAHs	$t_{1/2} \sim 10$ hr. for all PAHs
Benz[a]anthracene		10	0.45	370		
Dibenzanthracene [a]			0.42	340		

[a] Isomer not specified.

Source: Radding et al. (1976).

TABLE 5-19. PREDICTED HALF-LIVES FOR BENZO[a]PYRENE TRANSFORMATION AND REMOVAL PROCESSES IN GENERALIZED AQUATIC SYSTEMS

	Half-life (hours)			
Process	River	Eutrophic Pond	Eutrophic Lake	Oligotrophic Lake
Photolysis	3.0	7.5	7.5	1.5
Oxidation	>340	>340	>340	>340
Volatilization	140	350	700	700
Biodegradation	$>10^4$	$>10^4$	$>10^4$	$>10^4$
Hydrolysis	NA	NA	NA	NA

Source: Smith *et al*. (1978).

TABLE 5-20. BIOCONCENTRATION OF BENZO[a]PYRENE IN FRESHWATER AND SALTWATER SPECIES

Species	Duration	Bioconcentration Factor	Reference
		Freshwater Species	
Alga, *Oedogonium cardiacum*	3 days	5,258[a]	Lu *et al*. (1977)
Snail, *Physa* sp.	3 days	82,231[a]	Lu *et al*. (1977)
Cladoceran, *Daphnia pulex*	3 days	134,248[a]	Lu *et al*. (1977)
Mosquito, *Culex pipiens quinquefasciatus*	3 days	11,536[a]	Lu et al. (1977)
Mosquitofish, *Gambusia affinis*	3 days	930[a]	Lu *et al*. (1977)
		Saltwater Species	
Clam, *Rangia cuneata*	24 hours	8.66	Neff *et al*. (1976a)
Clam, *Rangia cuneata*	24 hours	236	Neff *et al*. (1976b)
Eastern oyster, *Crassostrea virginica*	14 days	242	Couch *et al*. (in press)
Mudsucker *Gillichthys mirabilis*	96 hours	0.048	Lee *et al*. (1972)
Tidepool sculpin, *Oligocottus maculosus*	1 hour	0.13	Lee *et al*. (1972)
Sand dab, *Citharichthys stigmacus*	1 hour	0.02	Lee *et al*. (1972)

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[a] Model ecosystem concentration factor.

concentration factors (BCFs) among species and over time, BaP accumulated to high levels (BCFs of 900-134,000) over 3 days in freshwater organisms, but to much lower levels (0.02-242 BCF) in marine biota in less than 3 days.

The rates of BaP uptake and depuration have been investigated in a microcosm study using freshwater insect larvae (midges), Daphnia, periphyton (diatoms), and bluegill sunfish. Organisms were exposed to C^{14}-labeled BaP at a concentration of 1.0 µg/l for 8 hours, and the rate of depuration was determined. In periphyton, accumulation was linear over time, surface-area dependent, and diffusion-limited. Daphnia had high uptake and depuration rates, perhaps due to their large surface area/volume ratio; very little biotransformation occurred in Daphnia, however. In the midges (Chironomus riparius), greater than 50% of the BaP was transformed into polar compounds in 1 hour, and 10% of the accumulated BaP was associated with the exo-skeleton. Bioconcentration factors derived for these insects were 950 (steady state) and 150 (based on BaP analysis). Half-lives of BaP in the four species tested ranged from 5.3-8.5 days (Leversee et al. 1980). From these and several other studies, there is evidence that considerable variation exists in uptake and depuration rates and in the levels found in various types of tissues, even with the same species and compound.

Several investigations of the accumulation of BaP by biota from sediment and water in the natural environment have been reported. Experiments on the uptake of hydrocarbons, including BaP, from contaminated sediments by marine organisms showed that deposit feeders (the clam Macoma inquinata and sipunculid Phascolosoma agassizii) generally accumulated the compound to a greater extent than the suspension feeder (the clam Protothaca staminea). Experiments with the deposit-feeding clam M. inquinata, however, indicate that compounds directly associated with particulate matter in sediment were less available for uptake than those released from sediment in the surrounding seawater. Concentration factors for uptake from sediment were ≤0.2, while those from seawater were 10-1349. This work indicated that the PAHs present in interstitial water may be a prime source of contamination for benthic infauna and probably accounted for much of the uptake by the sediment-feeding organisms (Roesijadi et al. 1978).

Numerous studies have examined the PAH content of aquatic organisms in relation to the environment in which the organisms reside. Data are presented in Table 5-21 that show the range of BaP levels in various species from different locations. In general, but not always, organisms sampled from coastal regions near major industrial or domestic point sources of PAH (e.g., Norfolk, VA harbor) contained higher BaP concentrations than organisms from more remote areas (Neff 1979). Benzo[a]pyrene concentrations in various aquatic organisms from several locations in the U.S. and southern Canada ranged from approximately <0.1 µg/kg (dry weight) to as high as 5000 µg/kg. In most cases, the organisms contained low (0-20 µg/kg range)

TABLE 5-21. CONCENTRATIONS OF BENZO[a]PYRENE IN TISSUES OF AQUATIC ORGANISMS

Organism	Location	BaP (μg/kg dry wt)	Reference
Cod (*Gadus* sp.)	Holsteinborg, Greenland	15	Mallet *et al*. (1963)
	Atlantic, 40 km off Toms River New Jersey, USA	<10	Pancirov and Brown (1977)
Menhaden	Raritan Bay, New Jersey, USA	6.0	Pancirov and Brown (1977)
Anchovy and smelt	San Diego Bay, California, USA	up to 5000	Lee *et al*. (1972)
Flounder (unidentified)	Long Branch, New Jersey, USA	<8	Pancirov and Brown (1977)
	South of Long Island, New York USA, 40°27'N 73°06'W	<4	Pancirov and Brown (1977)
Shrimp (*Penaeus aztecus*)	Palacios, Texas, USA	<4	Pancirov and Brown (1977)
Clam (unidentified)	Chincoteague, Virginia, USA	1.2	Pancirov and Brown (1977)
	Darien, Connecticut, USA	<0.4	Pancirov and Brown (1977)
Crab (unidentified)	Raritan Bay, New Jersey, USA	12	Pancirov and Brown (1977)
Crab (*Cellinectes sapidus*)	Chesapeake Bay, Virginia, USA	<2.0	Pancirov and Brown (1977)
Soft-shell clam (*Mya arenaria*)	Tillamook Bay, Oregon, USA	1.2	Mix *et al*. (1977)
	Alsea Bay, Oregon, USA	<0.4	Mix *et al*. (1977)
	Coos Bay, Oregon, USA	1.32-26.64	Mix *et al*. (1977)
Butter clam (*Saxidomas giganteus*)	Coos Bay, Oregon, USA	1.16- 4.20	Mix *et al*. (1977)
Oyster (*Crassostrea virginica*)	Norfolk Harbor, Virginia, USA	20-60	Cahnmann and Kuratsune (1957)
	Long Island Sound, USA	8.0	Pancirov and Brown (1977)
	Chincoteague, Virginia, USA	1.2	Pancirov and Brown (1977)
Crassostrea gigas	Tillamook Bay, Oregon, USA	<0.4	Mix *et al*. (1977)
Gaper clam (*Tresus capax*)	Tillamook Bay, Oregon, USA	0.64-12.8	Mix *et al*. (1977)
	Netarts Bay, Oregon, USA	<0.4	Mix *et al*. (1977)
	Yaquina Bay, Oregon, USA	1.0-2.24	Mix *et al*. (1977)
	Alsea Bay, Oregon, USA	0.4	Mix *et al*. (1977)
	Coos Bay, Oregon, USA	0.56-2.04	Mix *et al*. (1977)

TABLE 5-21. CONCENTRATIONS OF BENZO[a]PYRENE IN TISSUES OF AQUATIC ORGANISMS (Continued)

Organism	Location	BaP (µg/kg dry wt)	Reference
Mussel *Mytilus edulis*	Falmouth, Massachusetts USA		
	Little Sippewissett	<2	Pancirov and Brown (1977)
	Wild Harbor	2	Pancirov and Brown (1977)
	Tillamook Bay, Oregon, USA	<0.4-67.4	Mix *et al.* (1977)
	Yaquina Bay, Oregon, USA	0.48-120.8	Mix *et al.* (1977)
	Alsea Bay, Oregon, USA	<0.4	Mix *et al.* (1977)
	Vancouver, B.C., Canada		Dunn and Stich (1975)
	outer harbor	8 ± 1.2	
	wharf, marina and dock areas	72 ± 19.6	
	inner harbor	168 ± 24	
	Vancouver marina area		
	(May'74)	7.6 ± 0.4	
	(Sept '74)	7.2 ± 0.4	
	wharf area on rock and cables		
	(May '74)	52 ± 21.2	
	(Sept '74)	156 ± 29.2	
	creosoted pilings		
	(May '74)	272 ± 52	
	(Sept '74)	532 ± 76	
M. edulis and *M. californeanus*	25 stations between Bodega Head and San Diego, California, USA	<0.4-32.8	Dunn and Young (1976)
M. californeanus	West coast of Vancouver Island, British Columbia, Canada	0.4 ± 0.4	Dunn and Stich (1975)

BaP concentrations, except for animals collected from severely polluted areas or from the immediate vicinity of marina (creosoted pilings) or fish processing facilities (Mix et al. 1979). Fish from San Diego Bay, a large shipping port, contained up to 5000 μg/kg BaP (dry weight) (Lee et al. 1972).

Several species of bivalve mollusks (clams) from Oregon estuaries were sampled for levels of BaP. The maximum concentrations found were in the range of 15-30 ng/g (ppb). However, specimens from Coos Bay, the most heavily industrialized bay on the Oregon Coast, did not contain significant levels of BaP overall, except for the softshell calm *Mya arenaria* (Mix et al. 1977).

One report of BaP accumulation by mussels following an oil spill in a coastal area indicated that after 11 days, *Mytilus edulis* contained approximately 55 μg/kg BaP (Bories et al. 1976).

Biotransformation

The process of biotransformation and the resultant metabolites of BaP and other PAHs in aquatic organisms have been studied fairly extensively. It is known that enzymatic activity in the excretory systems -- i.e. hepatopancreas, intestine, etc -- of these organisms, facilitates the metabolizing of PAHs and excretion of water-soluble products. The extent to which animals are able to remove PAHs enzymatically from their tissues varies among species. Some marine organisms, primarily the invertebrates including starfish, sea anemones, oysters, and ctenophores, have been found to have very low or non-existent enzyme activity related to hydrocarbon metabolism. One explanation may be that organisms that pass large volumes of water across their tissues have not developed the enzyme mechanisms important in the toxification/detoxification system (Lee et al. 1977). Most of the teleost fish studied so far (including flounder, mackerel, trout, salmon, cunner, and sheepshead) and some members of the Phyla Arthropoda and Annelida are able to metabolize PAHs to polar compounds (Neff 1979). Such metabolic activity has not been detected in several species of marine algae (Payne 1977).

Microbial Biotransformation

Some of the members of the BaP group of PAHs are subject to microbial breakdown; on the whole, though, biodegradation is slower and not as extensive in this group as in the lower-molecular-weight PAHs. In a comparative study of the relative biodegradability of approximately 42 different polynuclear aromatic hydrocarbon compounds, McKenna and Heath (1976) found no significant biodegradation of compounds with more than three rings.

Within the BaP group, biodegradation rate data were available for only BaP, benz[a]anthracene, chrysene, and dibenz[a,h]anthracene. Within this group, the rate and extent of degradation is variable. Since structural factors such as ring number and molecular geometry influence biodegradation, there is danger in extrapolating from even relatively similar compounds to compounds for which no data are available. Other influences include environmental fate characteristics of the compounds, such as adsorption, solubility, vaporization, and other competitive transformation reactions (if any). Most of the biodegradation studies on PAHs are laboratory investigations, commonly conducted in simplified systems with the goal of eliciting biodegradation. Under environmental conditions, persistence may be longer than that measured in laboratory studies due to influences of external factors usually controlled for in the laboratory. As noted in Section 3.2.3.4, these environmental factors may include availability of oxygen, soil solution pH, and the presence of natural humic polymers.

Microorganisms act on PAHs by removing one cyclic unit at a time (Alexander 1977). This is supported by an observed inverse relationship between ring number and degradation rate.[1] Presumably a process analogous to that described for anthracene (Section 4.2.3.4) (Alexander 1977) occurs in the breakdown of the BaP group.

Presented in Table 5-22 are reported biodegradation products derived from the breakdown of two members of the group: BaP and benz[a]anthracene. The products are primarily intermediates in the multistep pathway of reactions leading to eventual complete mineralization. Gibson et al. (1975) isolated a mutant strain of the bacterium, Beijerinckia, which converted benz[a]anthracene to four isomeric dihydrodiols. (However, this study had limitations. For example, it was not possible to confirm whether the first step in biodegradation was removal of one ring from the molecule.) No other data were available on other group members.

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[1] It must be mentioned, however, that other factors such as substituent type and size, interfere with this relationship. Malaney did not find a direct relationship between ring number and biodegradability (Malaney et al. 1967).

TABLE 5-22. BIODEGRADATION PRODUCTS REPORTED FOR THE BENZO[a]PYRENE GROUP PAHs

PAH	Degradation Products
Benzo[a]pyrene[a]	cis-9,10-dihydroxy-9,10-dihydrobenzo[a]pyrene[b]
Benz[a]anthracene[a]	cis-1,2-dihydroxyl-1,2-dihydrobenzo[a]anthracene[b]

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[a] Fungi.

[b] Tentative identification.

Source: Gibson (1976).

The rate of biodegradation is quite variable for the BaP group of PAHs. Table 5-23 presents quantified rates of biodegradation reported for the BaP group in soil, freshwater, and estuarine systems. Due to the variety of test methods, analytical techniques, microbial species, and data analyses used in biodegradation testing, it is difficult to compare the results from the different tests reported in Table 5-23.

A total of nine studies was available on BaP; five of these studies found no degradation, two found minimal degradation, and two found relatively rapid degradation, as much as 86% in 5 days. Degradation of acclimated populations was generally faster than that of unacclimated ones. The addition of naphthalene, or especially phenanthrene, significantly increased biodegradation (McKenna and Heath 1976), apparently due to cometabolism. The results of five studies on benz[a]anthracene are similar to those found for BaP; in some cases no degradation occurred, but in one study as much as 41% was lost in 1 week. The addition of other PAHs again resulted in an increase in degradation. The one study available on chrysene found a moderate rate of decay, as much as 59% in 1 week. The sole study on dibenz[a]anthracene found negligible degradation for the compound alone, but a significantly higher rate in the presence of phenanthrene (McKenna and Heath 1976). No biodegradation data were available for acenaphthylene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[g,h,i]perylene, or ideno[1,2,3-c,d]pyrene.

A study on biodegradation of BaP and benz[a]anthracene in estuarine sediment populations found that the presence of a polychaete worm species increased the rate of degradation, possibly due to its role in sediment mixing or metabolism of the substances. Degradation was greatest in populations associated with large-grain-size sediment and higher in the surface than in subsurface sediment layers. Approximately 50% of the initial benz[a]anthracene concentration was degraded after 30 weeks in fine sand without the polychaete worm. No long-term measurements were reported for BaP (Gardner et al. 1979).

There is other evidence that BaP and benz[a]anthracene are not readily degradable by microbial populations. Biological treatment in wastewater treatment is reported to be ineffective at removing PAHs from waste streams (Andelman and Snodgrass 1974). Smith et al. (1978) were unable to isolate enrichment microbial cultures capable of degrading either compound; however, the authors allowed that under environmental conditions such microbes may exist.

The turnover time and transformation rate of PAHs in both acclimated and unacclimated stream populations (Schwall and Herbes 1978) provide a good estimation of the environmental persistence of these compounds, as well as the importance of microbial adaptation (see Table 5-24). The turnover time in acclimated populations ranged from 417 days for benzo[a]anthracene to more than 3.5 years for BaP. Turnover times in non-acclimated populations were significantly greater by at least an

TABLE 5-23. BIODEGRADATION RATES OF THE BENZO[a]PYRENE GROUP PAHs: INDIVIDUAL COMPOUND STUDIES

Test Type/Population Origin	Compound Tested	Results	Reference
Static flask (wastewater population)	Benz[a]anthracene	Inconsistent degradation over month period of acclimation from 0% degraded to 41% degraded in one week at 5 mg/l	Quave *et al.* (1980)
Static flask (wastewater population)	Chrysene	59% lost at 5 mg/l and 38% at 10 mg/l at one week in acclimated culture	Quave *et al.* (1980)
Freshwater populations - enrichment shake flask, also using naphthalene in culture	Benzo[a]pyrene	No degradation observed in 6-week period	Colwell and Sayler (1978)
	Benz[a]anthracene	No degradation observed in 6-week period	Colwell and Sayler (1978)
Adapted soil populations of *Pseudomonas aeruginosa* and *Escherischia coli*	Benzo[a]pyrene	90% taken up from medium, 10-26% metabolized	Lorbacher *et al.* (1971)
Salmonella typhimurium, *Aerobacter aerogenes*, *Escherischia coli*, *Saccharomyces cerevisiae*	Benzo[a]pyrene	Species accumulate compound but little metabolized. Can take up as much as 1 to 2 x 10^{-10} µg/cell (*E. coli*).	Moore and Harrison (1965)
Mycobacterium flavum *M. rubrum*, *M. lacticolum*, *M. smeginatis*, *Bacillus megaterium*, *Bacillus sphaericus*	Benzo[a]pyrene	*M. rubrum* and *M. flavum* metabolized 50% of compound in 4 days. Other species accumulated the compound (no mention of biodegradation)	Poglazova, *et al.* (1966, 1976a,b)

TABLE 5-23. BIODEGRADATION RATES OF THE BENZO[a]PYRENE GROUP PAHs: INDIVIDUAL COMPOUND STUDIES (Continued)

Test Type/Population Origin	Compound Tested	Results			Reference
Coastal estuary sediment populations (3 types) with and without presence of polychaete worm, *Capitella capitata*	Benzo[a]pyrene Benz[a]anthracene		% removed in 1 week		Gardner *et al.* (1979)
		Experiment	BaP	BaA	
		Fine sand	1.2	1.5	
		Fine sand & *C. capitata*	2.4	2.7	
		Med. sand	1.4	1.8	
		Med. sand & *C. capitata*	3.0	3.0	
		Marsh sed.	0.84	1.4	
		Marsh sed. & *C. capitata*	1.98	1.8	
Soil bacteria from benzopyrene contaminated area and from non-contaminated area	Benzo[a]pyrene	Acclimated population metabolized (75-86% of compound in 5 days; non-acclimated population 48-59% in same period			Shabad (1978) Shabad (1971a,b) Shabad *et al.* (1971b)
Bacteria in power plant and coke over wastewater	Benzo[a]pyrene	Metabolized <15% of compound			Poglazova *et al.* (1972)
$^{14}CO_2$ evolution with sea water population from treated area	benz[a]anthracene benzo[a]pyrene	Not degraded Not degraded			Lee *et al.* (1978)

TABLE 5-23. BIODEGRADATION RATES OF THE BENZO[a]PYRENE GROUP PAHs: INDIVIDUAL COMPOUND STUDIES (Continued)

Test Type/Population Origin	Compound Tested	Results		Reference
$CO_2{}^{14}$ evolution from contaminated stream sediment population	$[C^{14}]$ benz[a]anthracene	10^{-4}/h		Schwall and Herbes (1978)
	$[C^{14}]$ benz[a]pyrene	No measurable transformation in 26 days		
Shake flasks with natural water populations		Percent main compound (column 2) remaining at 4 weeks		McKenna and Heath (1976)
		+ naphthalene	+ phenanthrene	
	Benzo[a]pyrene	83.5	38.3	
	Benz[a]anthracene	58.3	33.8	
	Dibenz[a,h]anthracene	92.7	32.9	
		Negligible degradation was observed for each compound alone.		

TABLE 5-24. KINETIC PARAMETERS OF BIOTRANSFORMATION OF BENZ[a]ANTHRACENE AND BENZO[a]PYRENE

	Rate Constant k (1/h)		Turnover Time[a]		Transformation Rate (μg/g/hr)	
Compound	Contaminated	Uncontaminated	Contaminated	Uncontaminated	Contaminated	Uncontaminated
Benz[a]anthracene	1.0×10^{-4}	4.0×10^{-6}	417 days	28.5 years	1.2×10^{-5}	$<4 \times 10^{-8}$
Benzo[a]pyrene	$<3 \times 10^{-5}$	$<3 \times 10^{-5}$	3.5 years	57 years	$<2 \times 10^{-6}$	$<3 \times 10^{-7}$

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[a] Turnover rate = 1/k

[b] Transformation rate = k x concentration

Source: Schwall and Herbes (1978).

order of magnitude: an estimated 28.5 years and 57 years, respectively. How applicable these aquatic turnover times are for soil systems is unknown. However, the turnover time for the acclimated population could be comparable to that of a well-acclimated soil population.

In another small-scale biodegradation study using acclimated stream populations, the degradation time of PAHs increased 30-100 times for each additional ring from naphthalene to benz[a]anthracene (Schwall and Herbes 1978). In a static flask study of several PAHs (Quave *et al*. 1980), a considerable variation was observed over a four-ring range. Acclimation was required for all but benz[a]anthracene; for some compounds 10 mg/l of the compound was inhibitory (Quave *et al*. 1980).

Thus the benzo[a]pyrene group of PAHs is relatively resistant, as a class, to biodegradation, especially in comparison with lower-ringed aromatics. Most biodegradation studies are too short-term to quantify the half-lives for persistence of most of these PAHs. In some cases, half-lives on the order of weeks were reported; however, in general, PAHs with three rings or more were presistent on the order of months or longer. No information at all was available for acenaphthylene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[g,h,i]perylene, and ideno[1,2,3-c,d]pyrene.

5.2.3.3 Modeling of Environmental Distribution

Mackay Model

The Mackay equilibrium partitioning model (Mackay 1979) described in Section 3.2, was utilized for BaP. The values for the input parameters used in the Mackay model and the predicted distribution of BaP are given in Tables 5-25 and 5-26. Benzo[a]pyrene has a large adsorption coefficient for soil or sediment and a very low water solubility and vapor pressure. These characteristics suggest very little partitioning into the air or water with most of the BaP concentrated in the sediments. The differences in partitioning among the solid phases are a result of the relative amounts of each in the model ecosystem. The concentrations of material in the air and water compartments are significantly lower than those in the solid phases, while the concentration in the aquatic biota is somewhat higher. The number of moles predicted to reside in each compartment is directly proportional to the total number of moles in the whole system.

The Mackay model predicts that 99.9% of the BaP load to the system will reside in the sediment compartment. Less than 0.1% will be present in the water column, and less than 0.001% will partition to the atmosphere. Dibenz[a,h]anthracene, benz[a]anthracene, benzo[g,h,i]-perylene, indeno[1,2,3-c,d]pyrene all have physical properties similar to

TABLE 5-25. VALUES OF PARAMETERS USED FOR CALCULATING THE EQUILIBRIUM DISTRIBUTION OF BENZO[a]PYRENE PREDICTED BY THE MACKAY FUGACITY MODEL

Chemical-Specific Parameters (25°C):	
Henry's Law Constant (atm m^3/mole)	4.8×10^{-7}
Adsorption Coefficients:	
Suspended Solids	1.26×10^4
Sediment	1.26×10^4
Biota ($0.2 \times K_{ow}$)	2.2×10^5
Total Amount in System (kg)	11[a]
Compartment-Specific Parameter (25°C):	
Air	
area	1×10^4 m^2
depth	3×10^3 m
volume	3×10^2 m^3
Water	
area	1×10^4 m^2
depth	2m
volume	2×10^4 m^3
biomass content	129 mg/l
suspended solids	30 mg/l
Sediment	
area	1×10^4 m^2
depth	5×10^{-1} m
volume	5×10^3 m^3
biomass content	50.018/m^3
wet sediment density (sediment dry weight = (100 x wet weight)/137)	1.858/cm^3

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[a] The load to the MacKay model was assumed to be equal to the total pond accumulation predicted by EXAMS.

TABLE 5-26. EQUILIBRIUM PARTITIONING OF BENZO[a]PYRENE CALCULATED USING MACKAY'S FUGACITY MODEL

	Partitioning at Equilibrium		
Compartment	Moles	Concentration	Percent
Air	2.95×10^{-4}	2.48×10^{-4} µg/m^3	0.007
Water	1.02×10^{-2}	0.13 µg/l	0.0235
Suspended Solids	3.88×10^{-3}	1.628 mg/kg	0.0089
Sediment	43.65	1.628 mg/kg	99.89
Aquatic Biota	2.91×10^{-2}	28.38 mg/kg	0.0666
Biotic Sediment	2.82×10^{-3}	28.38 mg/kg	0.0065

those of benzo[a]pyrene, and would be expected to be partitioned into the various environmental compartments in the same manner. The benzofluoranthenes have larger vapor pressures and Henry's law constants, suggesting they might partition into air to a somewhat greater extent than the other compounds in this group. Acenaphthylene has physical properties more closely resembling naphthalene and anthracene, and atmospheric partitioning for this compound would be expected to be greater than for BaP.

EXAMS

The U.S. EPA EXAMS (Exposure Analysis Modeling System) model (U.S. EPA 1890b), described in Section 3.1 was run for BaP. All six EXAMS environments (pond, eutrophic lake, oligotrophic lake, river, coastal plain river and turbid river) were modelled using the ecosystem parameters that are provided with the model. The input parameters for BaP are given in Table 5-27. Any variables not in the table were set to zero, since these variables (such as hydrolysis) were generally not considered important for BaP. Photolysis is an important fate process for these PAHs, thus the absorption spectrum was included as input (Table 5-28).

A loading rate of 0.1 kg/hour was specified for the BaP calculations. However, in some environments this loading would cause a concentration of benzo[a]pyrene in the input stream that was greater than the maximum water solubility of the compound. When this type of overloading occurs, the EXAMS model automatically decreases the loading to a physically allowable level. Once the concentration of BaP is below the maximum solubility level, the maximum concentrations and accumulations are proportional to the loading, and the self-purification times and disposal rates remain unaffected. As shown in Table 5-29, the loading rates for the relatively static aquatic systems (ponds and lakes) were reduced by the model to 3.6×10^{-2} kg/day or lower. The load to the rivers was allowed to remain at 2.4 kg/day due to the fact that rapid dilution prevented BaP concentrations above solubility levels. The materials balance (Section 5.1) suggests that a maximum loading would be approximately 3×10^{-2} kg/day. Therefore, the concentration estimates for the pond and lakes are reasonable, but the actual river concentrations would be expected to be lower than the EXAMS estimates, shown in the following tables, by a factor of 80.

Table 5-29 summarizes the BaP concentrations predicted for the simulated environments under steady-state conditions. Concentrations in water are generally 10^2-10^4 times lower than concentrations in sediment and biota. The steady-state accumulation of BaP is highest in those environments with a higher biomass, i.e., coastal river, eutrophic lake and pond. The distribution and fate of BaP in the aquatic systems are presented in Table 5-30. In the oligotrophic lake, about 81% of the BaP load remains in the sediment, while more than 94% of the load in the rest of the environments resides in the sediment.

TABLE 5-27. INPUT PARAMETERS FOR EXAMS MODELING OF THE FATE OF BENZO[a]PYRENE IN GENERALIZED AQUATIC SYSTEMS

Explanation	Value
Molecular weight	252.0
Ratio volitilization: re-aeration rate	0.3760
Henry's Law constant	4.8×10^{-7}
Quantum yield in water(at 313 nm)	8.9×10^{-4}
Absorption spectra	See Table 5-28
Aqueous solubility(mg/l)	3.8×10^{-3}
Partition coefficient for biomass:H_2O	1.89×10^{5}
Octanol:H_2O partition coefficient	1.1×10^{6}
Second-order biolysis rate constant: in sediment and in the water column	3.0×10^{-12}
Increase in biolysis rate constant for 10°C increase in temperature	2.0

Source: SRI (1980).

TABLE 5-28. MOLAR ABSORBTIVITY FOR BENZO[a]PYRENE AS A FUNCTION OF WAVELENGTHS

Interval	Center of Interval (nm)	Molar Absorbtivity (1cm/mole/1)
1	297.5	46,600
2	300.0	27,700
3	302.5	13,900
4	305.0	6,670
5	307.5	4,840
6	310.0	3,970
7	312.5	3,870
8	315.0	3,650
9	317.0	3,730
10	320.0	3,570
11	323.1	3,650
12	330.0	5,400
13	340.0	8,330
14	350.0	12,300
15	360.0	18,100
16	370.0	19,680
17	380.0	21,910
18	390.0	15,160
19	400.0	2,100

Source: Smith *et al*. (1978).

TABLE 5-29. STEADY-STATE CONCENTRATIONS OF BENZO[a]PYRENE IN VARIOUS GENERALIZED AQUATIC SYSTEMS RESULTING FROM CONTINUOUS DISCHARGE[a]

		Maximum Concentrations						
System	Loading (kg/hr)	Water Dissolved (mg/l)	Water Total (mg/l)	Maximum in Sediment Deposits (mg/kg)	Plankton (µg/g)	Benthos (µg/g)	Total Steady-State Accumulation (kg)	Total Daily Load (kg/day)
Pond	6.2×10^{-5}	2.2×10^{-4}	1.2×10^{-3}	16	43	43	11	1.5×10^{-3}
Eutrophic Lake	1.5×10^{-3}	1.6×10^{-4}	1.7×10^{-3}	4.3	30	30	59	3.6×10^{-2}
Oligotrophic Lake	1.5×10^{-3}	3.2×10^{-5}	3.4×10^{-4}	0.11	6.1	0.3	0.047	3.5×10^{-2}
River	0.1	4.1×10^{-5}	1.5×10^{-4}	0.33	7.8	1.4	6.2	2.4
Turbid River	0.1	1.3×10^{-5}	1.2×10^{-4}	0.09	2.4	1.2	1.9	2.4
Coastal River	0.1	7.4×10^{-4}	1.9×10^{-3}	19	140	37	290	2.4

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[a] All data simulated by EXAMS (U.S. EPA 1980b) model (see text for further information).

TABLE 5-30. THE FATE OF BENZO[a]PYRENE IN VARIOUS GENERALIZED AQUATIC SYSTEMS[a]

	Percent Distribution		Percent Lost by Various Processes				
System	Residing in Water at Steady-State	Residing in Sediment at Steady-State	Transformed by Chemical Process	Transformed by Biological Process	Volatilized	Lost by Other Processes[b]	Time for System Self-Purification[c]
Pond	0.24	99.76	68.79	0	0.16	31.03	69 years
Eutrophic Lake	1.85	98.15	95.10	0.05	0.21	4.64	25 years
Oligotrophic Lake	18.75	81.25	99.97	0	0.02	0.01	393 days
River	2.17	97.83	0.04	0	0	99.96	95 days
Turbid River	5.63	94.37	0	0	0	100	89 days
Coastal River	0.56	99.44	2.89	0	0.02	97.08	376 days

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[a] All data simulated by the EXAMS (U.S. EPA 1980b) model. See text for further information.

[b] Including loss through physical transport beyond system boundaries.

[c] Estimate for removal of ca. 97% of the toxicant accumulated in system. Estimated from the results of the half-lives for the toxicant in bottom sediment and water columns, with overall cleansing time weighted according to the pollutant's initial distribution.

The ultimate fate of BaP in these systems is controlled primarily by chemical processes in the static systems, and physical transport beyond the system boundaries for the river systems. Volatilization and biological degradation are negligible processes for BaP.

The EXAMS estimates for the persistence of BaP upon cessation of loading in the aquatic environments are given in Table 5-31. The fastest system to purify itself of the BaP load is the turbid river: 8.93% is lost within one day: Table 5-30 indicates a self-purification time of 89 days due to physical transport. The other river systems show similar purification characteristics, although somewhat slower. Of the pond and lake systems, the oligotrophic lake exhibits the lowest persistence of BaP. Within 24 days 30% of the accumulated BaP will be lost. Chemical degradation processes control the fate of BaP in the oligotrophic lake, probably due to the high rate of photolysis in the clear waters and the fact that almost 20% of the BaP is residing in the water column and available for absorption of sunlight. The pond and eutrophic lake require much longer time periods for removal of BaP, and have self-purification times of 69 years and 25 years, respectively. Chemical transformation appears to account for the ultimate fate of BaP in these systems. However, the waters of the pond and eutrophic lake are not clear and photolysis will be slower than in the oligotrophic lake. Furthermore, more than 98% of the BaP resides in the sediment of these systems, where light penetration is extremely low.

Comparison of Mackay and EXAMS Models

The Mackay model used is simply a partitioning model, whereas EXAMS utilizes kinetic data to predict the fate of the compound after partitioning. Therefore, the models are not directly comparable. The EXAMS pond environment was taken to be the best system to compare with the Mackay model, since there is very little transport across system boundaries. The load to the Mackay model was assumed to be equal to the total pond accumulation predicted by EXAMS, 11 kg.

Table 5-32 summarizes the concentration and distribution data predicted by the two models. The percentages of distribution predicted by the two models agree quite well; the estimated concentrations are also in agreement within an order of magnitude. The most important conclusion from these data is that there will be very little BaP in the dissolved state, and most of the aquatic load will be removed to the sediment.

TABLE 5-31. THE PERSISTENCE OF BENZO[a]PYRENE IN VARIOUS GENERALIZED AQUATIC SYSTEMS AFTER CESSATION OF LOADING[a]

System	Time Period (days)	% Lost from Water	% Lost from Sediment	% Lost from Total System
Pond	1460	19.02	18.17	18.18
Eutrophic Lake	2190	71.93	56.26	56.55
Oligotrophic Lake	24	92.63	16.01	30.36
River	3.5	94.57	11.74	13.54
Turbid River	1	98.21	3.6	8.93
Coastal River	168	87.52	78.67	78.27

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[a] All data simulated by the EXAMS (U.S. EPA 1980b) model. See text for further information.

TABLE 5-32. COMPARISON OF RESULTS FROM MACKAY'S EQUILIBRIUM MODEL AND EXAMS FOR BENZO[a]PYRENE IN A POND SYSTEM

EXAMS Results (Pond: $1.5\text{x}10^{-3}$ kg/day loading, 11 kg steady-state accumulation)		Mackay Results (11 kg in system)	
	Maximum Concentrations		Concentrations
Water (Total)	0.2 μg/l	Water (dissolved)	0.13 μg/l
Water Biota	43 mg/kg	Suspended Solids	1.6 mg/kg
Sediment Biota	43 mg/kg	Water Biota	28.4 mg/kg
Sediment	16 mg/kg	Sediment Biota	28.4 mg/kg
		Sediment	1.6 mg/kg
	Steady-State Accumulation		Percent of Chemical per Compartment
% in Water	0.24	% in water[a]	0.1
% in Sediment	99.76	% in Sediment	99.9

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[a] Includes BaP dissolved in water, adsorbed on suspended solids, and in aquatic biota compartment.

5.2.4 Monitoring Data

PAHs, especially those in the BaP group, have been extensively monitored in environmental media. This section contains a discussion of the monitoring data contained in STORET, as well as a brief review of other data from the literature. In general, the most information is available for BaP, due to the emphasis on monitoring for this compound because of its carcinogenic nature.

5.2.4.1 STORET

Ambient and Effluent Water

STORET monitoring data for the benzo[a]pyrene group reflect sampling activities in twenty-nine states and the U.S. territory of Puerto Rico.

As Table 5-33 indicates, observations in STORET indicate low concentrations for the BaP group PAHs. Generally, levels are less than the most frequently used detection limit of 10 μg/l. For three of the compounds, benzo[g,h,i]perylene, dibenz[a,h]anthracene, and indeno-[1,2,3-c,d]pyrene, the detection limits most used during analysis were higher than 10 μg/l; however, levels in ambient waters do not exceed 100 μg/l. Table 5-34 presents the maximum detection limits used in monitoring and the ranges of unremarked observations[2] for each pollutant.

Table 5-33 shows the distribution of effluent concentrations, remarked and unremarked, for the benzo[a]pyrene group. Roughly 40% of the observations indicate detection limits equal to or less than 1 μg/l, and 50% with detection limits between 1.1 μg/l and 10 μg/l. The number of unremarked observations does not exceed eight for any one pollutant. Positive values range from less than 1 μg/l to over 1000 μg/l.

Sediment and Soil

Data summaries are presented in Tables 5-34 and 5-35 for concentrations in sediment for the benzo[a]pyrene group contained within the STORET data base. For each pollutant, roughly 90% of the observations are remarked. The distribution indicates that, of the remarked observations, detection limits equal to or less than 1 μg/kg (dry weight) are used about 30% of the time, detection limits between 100.1 and 1000 μg/kg are used 10% of the time, and detection limits over 1000 μg/kg are used 60% of the time. No more than a dozen unremarked observations are recorded for each pollutant, and these positive values are generally less than 1000 μg/kg.

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[2] Remarked observations are generally undetected values, unremarked observations are positive values.

TABLE 5-33. DISTRIBUTION OF OBSERVED AMBIENT AND EFFLUENT CONCENTRATIONS OF THE BENZO[a]PYRENE GROUP PAHs

	Number of Ambient Observations[a]											
	Remarked Data						Unremarked Data					
		Conc. (μg/1)						Conc. (μg/1)				
Compound	Total # Obs.	≤1	1.1-10	10.1-100	100.1-1000	>1000	Total # Obs.	≤1	1.1-10	10.1-100	100.1-1000	>1000
Benzo[a]pyrene	411	41	281	80	9		0					
Acenaphthylene	423	46	326	38	13		6	6				
Benz[a]anthracene	347	41	268	35	3		5	1	2		2	
Benzo[b]fluoranthene	236	41	154	41			0					
Benzo[k]fluoranthene	354	39	194	113	4	4	5				3	2
Benzo[g,h,i]perylene	407	37	128	230	8	4	0					
Chrysene	246	47	184	15			1	1				
Dibenz[a,h]anthracene	416	50	108	237	13	8	0					
Indeno[1,2,3-c,d]pyrene	404	45	121	222	12	4	0					

	Number of Effluent Observations[b]											
	Remarked Data						Unremarked Data					
		Conc. (μg/1)						Conc. (μg/1)				
	Total # Obs.	≤1	1.1-10	10.1-100	100.1-1000	>1000	Total # Obs.	≤1	1.1-10	10.1-100	100.1-1000	>1000
Benzo[a]pyrene	667	290	335	42			4	2	1			1
Acenaphthylene	575	196	362	17			8	4	4			
Benz[a]anthracene	559	190	352	17			4		1			3
Benzo[b]fluoranthene	512	215	287	10			2		1			1
Benzo[k]fluoranthene	659	325	320	14			2	2				
Benzo[g,h,i]perylene	677	296	305	76			3	2				1
Chrysene	508	188	315	5			7	2	1	1		3
Dibenz[a,h]anthracene	564	180	305	73	6		2			1	1	
Indeno [1,2,3-c,d]pyrene	666	282	303	81			6	5			1	

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[a] Data as of December 1, 1980, except data for Dibenz[a,h]anthracene, which are as of November 20, 1980.

[b] Data as of April 9, 1980.

Source: U.S. EPA (1980c).

TABLE 5-34. NUMBER, DETECTION LIMITS, AND RANGES OF OBSERVED CONCENTRATIONS IN AMBIENT WATER AND SEDIMENT FOR THE BENZO[a]PYRENE GROUP PAHs--STORET, 1980[a]

	Benzo[a]-pyrene	Acenaphthylene	Benz[a]-anthracene	Benzo[b]-fluoranthene	Benzo[k]-fluoranthene	Benzo-[g,h,i]-perylene	Chrysene	Dibenz[a,h]anthracene	Indeno-[1,2,3-c,d]-pyrene
Ambient Water (μg/l)									
Total No. Observations	411	429	352	236	359	407	247	416	404
Unremarked	0	6	5	0	5	0	1	0	0
Remarked	411	423	347	236	354	407	246	416	404
Maximum Detection Limit	800	640	400	25	1000	1600	25	1600	1600
Range of Unremarked Observations		0.01-0.12	1-400		320-1500		0.02		
Sediment (μg/kg--dry weight)									
Total No. Observations	125	125	116	109	121	123	102	130	117
Unremarked	11	12	12	13	12	5	11	2	5
Remarked	114	113	104	96	109	118	91	128	112
Maximum Detection Limit	10000	10000	10000	10000	10000	10000	10000	10000	10000
Range of Unremarked Observations	0.02-1400	0.002-93	6.2-340	3.4-310	0.9-1300	4.3-40.9	0.06-120	15.7-2600	1-35.8

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[a] Data as of November 20, 1980, except for Dibenz[a,h]anthracene, which are as of December 1, 1980.

Source: U.S. EPA (1980c).

TABLE 5-35. DISTRIBUTION OF OBSERVED SEDIMENT AND TISSUE CONCENTRATIONS OF THE BENZO[a]PYRENE GROUP PAHs

Number of Sediment Observations

	Remarked Data						Unremarked Data					
	Total # Obs.	Conc. (µg/kg-dry wt.) ≤1	1.1-10	10.1-100	100.1-1000	>1000	Total # Obs.	Conc. (µg/kg-dry wt.) ≤1	1.1-10	10.1-100	100.1-1000	>1000
Benzo[a]pyrene	114	30			19	65	11	2	1	5	2	1
Acenaphthalene	113	28		2	18	65	12	1	6	5		
Benz[a]anthracene	104	27			16	61	12	3	2	5	2	
Benzo[b]fluoranthene	96	28		1	17	50	13		2	2	9	
Benzo[k]fluoranthene	109	28			21	60	12	1	2	4	4	1
Benzo[g,h,i]perylene	118	35		1	12	70	5		3	2		
Chrysene	91	28			13	50	11	1	2	6	2	
Dibenz[a,h]anthracene	128	36			18	74	2			1		1
Indeno[1,2,3-c,d]pyrene	112	29		1	16	66	5	1	2	2		

Number of Tissue Observations

	Remarked Data						Unremarked Data					
	Total # Obs.	Conc. (mg/kg-wet wt.) ≤1	1.1-10	10.1-100	100.1-1000	>1000	Total # Obs.	Conc. (mg/kg-wet wt.) ≤1	1.1-10	10.1-100	100.1-1000	>1000
Benzo[a]pyrene	72	10	62				0					
Acenaphthylene	72	12	60				1	1				
Benz[a]anthracene	72	10	62				0					
Benzo[b]fluoranthene	72	10	62				0					
Benzo[k]fluoranthene	72	10	62				0					
Benzo[g,h,i]perylene	73	9	56	8			0					
Chrysene	68	9	58			1	2	2				
Dibenz[a,h]anthracene	73	9	55	9			0					
Indeno[1,2,3-c,d]pyrene	70	10	51	9			0					

Source: U.S. EPA (1980c).

Fish Tissue

As shown in Table 5-35, most concentrations for the benzo[a]pyrene group in fish tissue are remarked. The exceptions are observations for acenaphthylene, with one unremarked observation no higher than 1 mg/kg (wet weight), and chrysene, with two unremarked observations no higher than 1 mg/kg. The distribution indicates that, in general, roughly 15% of the time fish tissue samples are analyzed with detection limits not exceeding 1 mg/kg, and 85% of the time the detection limits are between 1.1 mg/kg and 10 mg/kg-wet wt.

5.2.4.2 Data From Other Sources

Ambient and Effluent Water

White and Vanderslice (1980) have thoroughly reviewed literature reports of concentrations of PAHs in ambient waters. The results of their review are presented as frequency distributions of reported concentrations in Table 5-36. The data show that levels are almost always less than 1 μg/l, and commonly less than 0.01 μg/l, suggesting that actual levels are probably much less than the 10 μg/l detection limit generally reported in the STORET data base.

A 1978 study by Basu and Saxena (1978) reports concentrations of BaP and of total PAHs in river waters that serve as municipal drinking water supplies. Their results are summarized in Table 5-37. The higher levels found in Monongahela probably result from coking operations in the area.

Andelman and Snodgrass (1974) have reported BaP concentrations of 2-300 μg/l in aqueous effluents from industrial operations such as shale oil recovery, oil refining coke byproduct recovery, and acetylene manufacture. The fact that the upper end of this range exceeds the BaP water solubility suggests that total (dissolved plus suspended) PAHs were being analyzed.

Davies et al. (1976) reported that levels of BaP group PAHs in the spent scrubber water of a British municipal refuse incinerator were on the order of 0.03-0.64 μg/l. These direct aqueous discharges represented only a small (< 1%) percentage of the total estimated PAH emissions from this facility, compared with stack gas (approximately 10%) and ash (approximately 90%) emissions of PAHs.

A systematic study of sources of priority pollutants to POTWs in Cincinnati, St. Louis, Atlanta, and Hartford (Levins et al. 1980) reported no detectable levels of BaP or other PAHs in this group in raw wastewater from residential, commercial, or industrial urban areas, at 4-10 μg/l detection limits.

Drinking Water

Basu and Saxena (1978) investigated levels of BaP group PAHs in several United States municipal drinking water supplies. The results

TABLE 5-36. CONCENTRATIONS OF BENZO[a]PYRENE GROUP PAHs IN AMBIENT WATER

Sample/PAH	Total No. of Observations	No. Observations in Indicated Concentrations (µg/l) Range					
		0.0001-0.001	0.001-0.01	0.01-0.1	0.1-1	1-10	> 10
River Water							
Benzo[a]pyrene	17	2	5	4	5		1
Benz[a]anthracene	5			4	1		
Benzo[b]fluoranthene	5		1	3	1		
Benzo[k]fluoranthene	12	1	2	7	2		
Benzo[g,h,i]perylene	13		3	6	4		
Chrysene	1			1			
Indeno(1,2,3-c,d)pyrene	12		2	6	4		
Lake Water							
Benzo[a]pyrene	4	1	1		1	1	
Benzo[b]fluoranthene	1	1					
Benzo[k]fluoranthene	1		1				
Benzo[g,h,i]perylene	1		1				
Groundwater							
Benzo[a]pyrene	6	3	1		2		
Benz[a]anthracene	2	1	1				
Benzo[b]fluoranthene	4	2	2				
Benzo[g,h,i]perylene	4	2	2				
Precipitation							
Benzo[a]pyrene	2			2			
Benz[a]anthracene	2			1	1		
Benzo[b]fluoranthene	2			1	1		
Benzo[k]fluoranthene	2			2			
Benzo[g,h,i]perylene	2			1	1		

Source: White and Vanderslice (1980).

TABLE 5-37. LEVELS OF PAHs IN THE MONONGAHELA, OHIO, AND DELAWARE RIVERS

River	Concentration (ng/l)	
	BaP	Total PAH
Monongahela, PA	42-77	600-660
Ohio, WV	5.6	58
Delaware, PA	4.1	350

Source: Basu and Saxena (1978).

reported for tap water in selected locations in New York, Pennsylvania, West Virginia, and Louisiana are given in Table 5-38. The highest value reported in this survey was 0.004 μg/l for benzo[g,h,i]perylene. However, most values were less than 0.001 μg/l.

Levins *et al*. (1980) reported no detectable levels of the BaP group PAHs in tap water samples from Cincinnati, St. Louis, Atlanta, and Hartford (detection limit of 4-10 μg/l).

Sediment and Soil

Heit (1979) has reported BaP concentrations of 6-305 μg/g (dry weight) in sediment samples collected at depths ranging from 0-55 cm from a lake near Los Angeles, CA. The largest concentration corresponds to a sample taken at a depth of 27-29 cm; this value was almost a factor of 10 above the concentrations at the other depths. Samples of other western lake sediments from rural areas showed BaP levels generally below the 2 μg/g detection limit in this study.

Giger and Blumer (1974) and Hites *et al*. (1977) have documented the BaP levels in marine sediments in the vicinity of the 1969 oil spill near West Falmouth, MA. BaP concentrations of 370 μg/kg and 340 μg/kg dry weight of sediment were observed in 1969 and 1970, respectively. Sediment core samples showed apparent historical levels of 380 μg/kg (in 1900) and 26 μg/kg (in 1850). By contrast, a parallel examination of Charles River, MA, sediment by Hites *et al*. (1977) showed a BaP level of 8000 μg/kg dry weight of sediment.

A research group at Research Triangle Institute (White and Vanderslice 1980) has reviewed reported data on PAH levels in soils. The results of that review for the BaP group PAHs are summarized in Table 5-39.

Fish Tissue

Shellfish tissue taken from estuarine waters indicated a higher level of BaP than those taken from marine environments (Pancirov and Brown 1977, Mix *et al*. 1976). Table 5-40 summarizes readily available data on the BaP group PAHs in edible marine organisms from a number of U.S. locations.

Air

Benzo[a]pyrene has been monitored in air in urban and industrial areas. Table 5-41 summarizes concentration ranges for BaP and related PAHs in U.S. urban air as cited in the U.S. EPA Criteria Document (U.S. EPA 1980a). Table 5-42 presents some earlier data, also from the Criteria Document, that illustrates the variability of PAH levels in different cities. Table 5-43 presents the frequency distribution of observations of BaP group PAHs in urban air, based upon the compilation of White and Vanderslice (1980).

TABLE 5-38. LEVELS OF BENZO[a]PYRENE GROUP PAHs IN DRINKING WATER

Location of Water Supply System	Raw Water Source	Sampling Date	Concentration data for Treated Drinking Water[a] (ng/l)			
			Benzo[a] pyrene	Benzo[k] fluoranthene	Benzo[g,h,i] perylene	Indeno[1,2,3-c,d] pyrene
Syracuse, NY	Lake Skaneatelas (uncontaminated)	12/16/76	0.3	0.4	0.4	---
Buffalo, NY	Lake Erie (contaminated with industrial discharge)	12/26/76	0.2	---	0.7	---
Pittsburgh, PA	Monongahela River (contaminated with coke oven effluent)	1/19/77	0.4	0.2	0.7	1.2
Huntington, WV	Ohio River (downstream from coke oven plants)	1/20/77	0.5	0.2	2.5	1.2
Endicott, NY	Groundwater (uncontaminated)	2/22/77	0.2	---	2.9	0.7
Hammondsport, NY	Keuka Lake (contaminated with agricultural waste)	2/28/77	0.3	0.1	1.9	0.9
Philadelphia, PA	Delaware River (contaminated with municipal waste)	3/5/77	0.3	---	4.0	1.7
Unidentified	Uncontaminated upland water	3/17/77	0.5	0.7	1.8	2.2
Lake George, NY	Lake George (contamination from recreational sources)	3/26/77	0.3	0.1	2.6	0.9
New Orleans, LA	Mississippi River(contaminated with industrial discharges)	5/1/77	1.6	0.6	---	---

[a] Data are for single analysis of one water sample taken from each of ten finished water supplies.

Source: Basu and Saxena (1978).

TABLE 5-39. FREQUENCY OF OBSERVATIONS OF BENZO[a]PYRENE GROUP PAHs IN SOIL AND SEDIMENT

Sample/PAH	Total No. of Observations	No. Observations in Indicated Concentration (μg/kg) Range						
		0.01-0.1	0.1-1.0	1-10	10-100	100-1000	1000-10,000	10,000-100,000
Rural Soil								
Benzo[a]pyrene	52	3	3	19	23	4		
Benz[a]anthracene	3			3				
Benzo[b]fluoranthene	3				3			
Benzo[k]fluoranthene	3			2	1			
Benzo[g,h,i]perylene	6			3	3			
Indeno[1,2,3-c,d]pyrene	3			2	1			
Urban Soil								
Benzo[a]pyrene	23			1	7	7	7	1
Benz[a]anthracene	2					1		1
Benzo[k]fluoranthene	2					1	1	
Benzo[g,h,i]perylene	2					1		1
Chrysene	2						1	1
Marine Sediments								
Benzo[a]pyrene	17				3	4	6	
Benz[a]anthracene	2			4	1	1		
Benzo[g,h,i]perylene	2				1	1		
Chrysene	2				1	1		
Soil Near Industrial Sources								
Benzo[a]pyrene	13				4	3	2	4

Source: White and Vanderslice (1980).

TABLE 5-40. REPORTED LEVELS OF BENZO[a]PYRENE GROUP PAHS IN EDIBLE MARINE ORGANISMS

		Concentration (μg/kg wet weight)		
Location of Sample	Marine Tissue	Benz[a]-anthracene	BaP	Other PAHs[a]
Long Island Sound	Oyster	8	2	<2-15
Chincoteague, VA				
Black Point	Oyster	0.1	0.2	<0.5-0.7
Little Toms Cove	Clam	0.3	0.3	<0.1-0.9
Darien, Conn. Scotts Cove	Clam	1	<1	1-3
Fish Market, Linden, NJ	Clam	<1	<1	<0.5-<3
Chesapeake Bay	Crab	<1.5	0.5	<0.5-<1.2
Raritan Bay	Crab	2	3	1-2
	Menhaden	<0.3	1.5	<0.3-1
Atlantic Ocean				
Long Branch, NJ	Flounder	<1	2	<1-<2
S. of Long Island	Flounder	<1	1	1
Falmouth, MA				
Little Sippewisset	Mussel	<0.2	<0.5	<0.2-0.3
Wild Harbor	Mussel	<0.6	<0.5	<0.5-1.2
Palacios, Texas	Shrimp	<0.2	<1	<0.3-<1 <0.5-<5
Atlantic Ocean				
25 mi. off Toms River, NJ	Codfish	<2	<1	<0.5-<1.5

Source: Pancirov and Brown 1977.

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[a] Includes chrysene, benzo[b]fluoranthene, benzo[e]pyrene, perylene, and benzo[g,h,i]perylene.

Source: Pancirov and Brown (1977).

TABLE 5-41. REPORTED LEVELS OF BENZO[a]PYRENE GROUP PAHs IN AIR OF U.S. CITIES

Compound	Concentration Range (ng/m^3)[a]	Reference
Benz[a]anthracene	0.18-4.6	Fox and Staley (1976), Gordon (1976)
Benzo[b]fluoranthene	0.1-1.6	Gordon and Bryan (1973)
Benzo[k]fluoranthene	0.18-5.2	Fox and Staley (1976)
Benzo[a]pyrene	0.13-3.2	Colucci and Begeman (1971)
Indeno[1,2,3-c,d]pyrene	0.03-1.34	Gordon (1976), Gordon and Bryan (1973
Chrysene	0.2-6.4	Gordon and Bryan (1973)
Benzo[g,h,i]perylene	0.2-9.2	Gordon and Bryan (1973)

[a] The concentration data for air can be converted to ppt (parts per trillion) by multiplying the value in ng/m^3 by the following factors (F=24.47/M.W.): benz[a]anthracene and chrysene, F=0.107; benzo[a]pyrene and benzofluoranthenes, F=0.097; indeno[1,2,3-c,d]pyrene and benzo[g,h,i]-perylene, F=0.089.

TABLE 5-42. CONCENTRATIONS OF BENZO[a]PYRENE GROUP PAHs IN THE AIR OF SELECTED U.S. CITIES, AVERAGE OF SUMMER AND WINTER VALUES

City	Concentration (ng/m^3)[a]		
	Benzo[a]pyrene	Benzo[k]fluoranthene	Benzo[g,h,i]perylene
Atlanta, GA	4.5	3.7	7.0
Birmingham, AL	15.7	8.8	13.2
Detroit, MI	18.5	12.5	21.3
Los Angeles, CA	2.9	3.1	10.2
Nashville, TN	13.2	8.0	10.2
New Orleans, LA	3.1	2.9	6.0
San Francisco, CA	1.3	1.0	5.1
Range	1.3-18.5	1.0-12.5	5.1-21.3
Mean	8.5	5.7	10.4

[a] Concentration data for air samples can be converted to ppt (parts per trillion) by multiplying the value in ng/m^3 by the following factors (F=24.47/M.W.): benzo[a]pyrene and benzo[k]fluoranthene, F=0.097; benzo[g,h,i]perylene, F=0.089.

Source: Sawicki *et al*. (1962).

TABLE 5-43. FREQUENCY OF AMBIENT CONCENTRATIONS OF BENZO[a]PYRENE GROUP PAHs IN URBAN AIR

PAH	Total No. of Observations	No. Observations in Indicated Concentration (ng/m^3) Range					
		0.01-0.1	0.1-1	1-10	10-100	100-1000	> 1000
Benzo[a]pyrene	116	1	6	45	54	9	1
Benz[a]anthracene	24	4	5	8	5	2	
Benzo[b]fluoranthene	8		4	4			
Benzo[k]fluoranthene	29	3	7	13	6		
Benzo[g,h,i]perylene	54	1	6	21	24	2	
Chrysene	12	1	2	6	2	1	
Indeno[1,2,3-c,d]pyrene	2		1	1			

Source: White and Vanderslice (1980).

Air samples collected in areas surrounding combustion sources show the highest levels of BaP in air. Table 5-44 presents some data showing that oil refineries, for example, can be associated with air levels of BaP 30 times those near airports. Katz and Chan (1980) note that an earlier U.S. EPA study found BaP levels in urban air to be 1.42 to 3.34 times higher in cities with coke oven facilities than in cities without coke ovens. Table 5-42 shows relatively high ambient levels of BaP in cities such as Detroit and Birmingham, compared with San Francisco, for example. These differences are due probably in considerable part to the different degrees of industrialization, although meteorologic and climatologic factors are also important.

Rural areas have been shown to have detectable levels of PAHs in ambient air, though the levels are well below any levels measured in heavily populated or industrial areas (U.S. EPA 1980a). Table 5-45 presents a frequency distribution for reported concentrations in rural air compiled by White and Vanderslice (1980).

Moschandreas _et al_. (1980) examined BaP levels in a few locations, indoors and outdoors, on woodburning and non-woodburning days. In the Boston metropolitan area, they found mean levels for various residences of 0.4-1.1 ng/m^3 indoors, and 0.4-0.9 ng/m^3 outdoors. On woodburning days in one residence, the mean level indoors was 4.7 ng/m^3, and the mean level outdoors was 1.3 ng/m^3. These levels suggest that there is little difference between indoor and outdoor concentrations of BaP except on days on which wood is being burned.

In order to place the ambient air data in perspective, Table 5-46 presents some data on PAHs in mainstream cigarette smoke (Severson _et al_. 1976). Note that these data are in μg/m^3 _vs_. ng/m^3 for ambient air concentrations and thus generally at least an order of magnitude higher.

5.2.5 Summary - Ultimate Fate and Distribution

Benzo[a]pyrene and the other PAHs in this group are released to the environment primarily as products of combustion. Virtually all of the BaP in the atmosphere is adsorbed onto airborne particulates. Even though photolysis of BaP in the atmosphere is expected to be rapid, wet and dry deposition of these PAHs are estimated to be the major input pathways to the aquatic environment. On the basis of transport model calculations, it was estimated that ∿44 kkg/yr BaP may be deposited on the surface of the U.S.; only a fraction of that will be introduced directly or indirectly to aquatic systems.

The relative composition of the PAH mixtures released from high-temperature combustion sources indicates that production of the unsubstituted, parent compound is favored. The fact that, within a PAH series, unsubstituted PAHs are the most abundant homologs in sediments from industrial areas (despite their higher solubility) further supports the conclusion that combustion is the major source of PAHs in the environment.

TABLE 5-44. RELATIONSHIP BETWEEN CONCENTRATION OF BENZO[a]PYRENE IN AIR AND DISTANCE FROM EMISSION SOURCE

Concentration of BaP (μg BaP/kg of particulate material)

Location	Distance from Source (m)				
	At Source	50	51-250	251-500	501-1500
Airport[a] - USSR	400	64	46	17	1.3
Oil refinery - USSR	12,000			1,200	120
Highway[b] (in town)	176	100	6	21	
(rural)	120			15	5

[a] Maximum values

[b] Total POM μg/kg

Source: White and Vanderslice (1980).

TABLE 5-45. FREQUENCY OF AMBIENT CONCENTRATIONS OF BENZO[a]PYRENE GROUP PAHs IN RURAL AIR

PAH	Observations	Number of Observations in Indicated Concentration(ng/m^3Range				
		0.001-0.01	0.01-0.1	0.1-1	1-10	10-100
Dibenz[a,h]anthracene	2		1	1		
Benzo[a]pyrene	16	2	2	2	6	6
Benzo[g,h,i]perylene	10				1	1

Source: White and Vanderslice (1980).

TABLE 5-46. CALCULATED CONCENTRATIONS OF BENZO[a]PYRENE AND RELATED PAHs IN MAINSTREAM CIGARETTE SMOKE

	Concentration ($\mu g/m^3$)		
PAH[a]	Non-Filter Cigarette	Filter Cigarette	"Low Tar" Cigarette (10 mg)
Benzo[a]pyrene plus Benzo[e]pyrene	85-86	28-32	16
Benzo[a]anthracene plus Chrysene plus Triphenylene	310-680	96-210	73

[a] Analysis method used did not give complete resolution of the PAHs; data are for partially resolved mixtures of PAHs.

Source: Calculated from data of Severson *et al*. (1976).

The Mackay equilibrium partitioning model was run for BaP, and the calculations indicate that 99.9% of the environmental BaP will reside in the sediment compartment; very small amounts will be found in the air (vapor phase BaP), water, or biota. The U.S. EPA EXAMS model was used to predict the fate and distribution of BaP in six generalized aquatic systems. The results indicate that in all of the systems modelled, maximum BaP concentrations in sediment can be expected to be higher than total water concentrations by factors up to 10^4. Aqueous BaP concentrations were estimated to range from 0.1 μg/l to 1.9 μg/l; sediment concentrations predicted by EXAMS range from 90 μg/kg to 1.9×10^4 μg/kg. The EXAMS data also indicate that more than 90% of the BaP will reside in the sediment for all aquatic systems examined, with the exception of the oligotrophic lake (81%).

The most significant fate processes for BaP in aquatic systems include adsorption (with subsequent transport to the sediment), and chemical degradation (photolysis). Volatilization and biodegradation, with half lives on the order of a number of days to months, are expected to be slow processes for BaP and the other PAHs in this group. The EXAMS calculations indicate that the relative importance of the various fate processes is determined by the actual conditions characteristic of the environmental systems. In the static pond and lake systems, chemical degradation will be the dominant fate process. However, in the more dynamic river systems, physical transport of BaP downstream is largely responsible for determining the ultimate fate. The persistence of BaP in aquatic systems is reflected in the self-purification times predicted by EXAMS. The times estimated for removal of 97% of the BaP accumulated in the turbid river system is 89 days, whereas the self purification time predicted for the static pond system is as high as 69 years.

Figure 5-9 gives a representation of the major inputs of BaP to the aquatic environment, as well as the dominant fate and transport pathways.

BaP has been extensively monitored in environmental media; there are fewer data for the other PAHs in this group. Most of the monitoring data in the STORET data base for the PAHs in the BaP group are remarked, i.e., reported to be below the detection limits. The PAH concentrations actually reported for sediment samples range from 0.002 μg/kg to 2600 μg/kg. Ambient water concentrations recorded in STORET range from 0.01 μg/l to 1500 μg/l. However, since sediment and aqueous samples were not taken from the same locations, very few conclusions can be based entirely on the limited data in STORET.

Data from other sources indicate that BaP concentrations in ambient waters are generally less than 1 μg/l; concentrations of BaP in industrial effluents were reported to be as high as 300 μg/l. Concentrations of BaP in drinking water were reported to range from 0.2 ng/l to 1.6 ng/l; the highest BaP concentration was in New Orleans, LA. In this survey, the highest concentration for the PAHs in the BaP group was 4.0 ng/l, reported

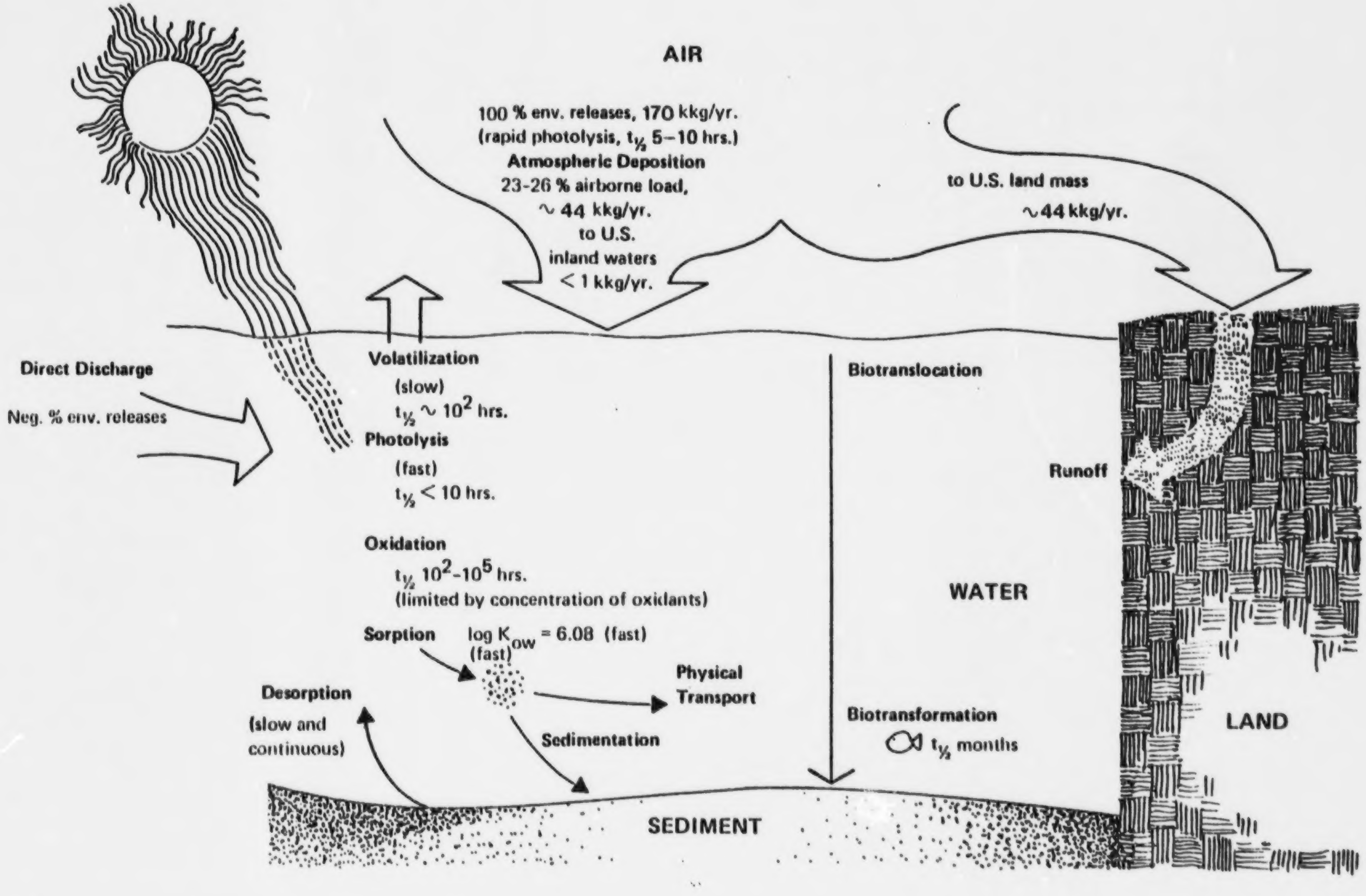

AIR
100 % env. releases, 170 kkg/yr.
(rapid photolysis, t½ 5–10 hrs.)
Atmospheric Deposition
23-26 % airborne load,
∼ 44 kkg/yr.
to U.S.
inland waters
< 1 kkg/yr.
to U.S. land mass
∼44 kkg/yr.
Direct Discharge
Neg. % env. releases
Volatilization
(slow)
t½ ∼ 10² hrs.
Photolysis
(fast)
t½ < 10 hrs.
Oxidation
t½ 10²-10⁵ hrs.
(limited by concentration of oxidants)
Sorption
log Kow = 6.08 (fast)
(fast)
Physical Transport
Sedimentation
Desorption
(slow and continuous)
Biotranslocation
Biotransformation
t½ months
Runoff
WATER
LAND
SEDIMENT

FIGURE 5–9 SOURCES AND FATE OF BENZO[a] PYRENE IN THE AQUATIC ENVIRONMENTS

for benzo[g,h,i]perylene in a drinking water sample taken from Philadelphia, PA. Benzo[a]pyrene has been reported at concentrations up to 300,000 μg/kg (dry weight) in lake sediment samples. In contrast, a Charles River, MA sediment sample showed a BaP level of 8000 μg/kg dry weight. Concentrations of these PAHs in urban soil were reported to be about two orders of magnitude higher than concentrations in rural soils.

Ambient air concentrations of these PAHs were also reported to be significantly higher in urban areas than in rural areas. In a study of seven urban areas, the highest PAH concentrations in air were reported for Detroit, MI, up to 21.3 ng/m^3 benzo[g,h,i]perylene, probably due to the high degree of industrialization in that city. Data presented in a separate review of ambient levels of PAHs indicate that most rural air concentrations are less than 10 ng/m^3; urban air concentrations up to 100 ng/m^3 were reported for many of these PAHs.

5.3 HUMAN EFFECTS AND EXPOSURE

5.3.1 Human Toxicity

5.3.1.1 Introduction

Data on the effects of ingestion of individual polycyclic aromatic hydrocarbons included in the benzo[a]pyrene group (i.e., acenaphthylene, benz[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[g,h,i]perylene, chrysene, dibenz[a,h]anthracene, indeno-[1,2,3-c,d]pyrene) on man or laboratory animals are few. We have concentrated our efforts on benzo[a]pyrene (BaP), the prototype PAH carcinogen since regulatory action to reduce levels of BaP will also result in the simultaneous reduction of other PAHs as well as noncarcinogenic compounds, and BaP is a major source of exposure.

5.3.1.2 Pharmacokinetics

There are no pharmacokinetic data for this group of PAHs in humans. Considerable animal data are available, however, for BaP from which generalizations can be drawn for other PAHs in this group.

Absorption and Distribution

Animal studies indicate that BaP is readily transported across the intestines, primarily by passive diffusion (Rees *et al*. 1971). It is also easily absorbed through the lungs (Kotin *et al*. 1969, Vainio *et al*. 1976). Rodent studies indicate wide tissue distribution following absorption of BaP (USEPA 1980). In rats, BaP disappears from blood and liver very rapidly following intravenous administration; the halftime for BaP disappearance from liver is about 10 minutes. This rapid elimination phase is followed by a slower disappearance phase lasting 6 hours or more in which the concentration of BaP increases in body fat and fatty tissues (e.g., breast) (Schlede *et al*. 1970). Since BaP has been shown to induce microsomal enzyme activity, prior exposure can accelerate both the rate of disappearance from tissues and excretion of metabolites into bile.

Metabolism

Most tumorigenic PAHs in themselves are not direct carcinogens but require metabolic activation before biological activity such as PAH-induced carcinogenesis can be expressed. Current understanding of the mechanisms involved in this process (Yang *et al*. 1978; Sims 1976, Lehr *et al*. 1978, Selkirk *et al*. 1975a,b Jerina *et al*. 1977a,b, Selkirk 1977) is as follows: PAHs are metabolized by the cytochrome P-450 dependent microsomal mixed function oxidase (MFO) system (often designated as the aryl hydrocarbon hydroxylase system). This enzyme system is readily inducible by exposure to a variety of chemicals and is found in most adult as well as fetal mammalian tissues, most especially in the liver. [Hepatic microsomal activity, although only a few percent of adult levels, has been detected in human fetuses as early as 6-7 weeks of gestation (Pelkonen 1976)]. The MFO catalyze the formation of reactive epoxide intermediates which are capable of forming covalent bonds with cellular constituents (DNA, RNA, proteins), and ultimately result in tumor formation. BP7,8-diol-9,10-epoxide is currently suspected to be the ultimate carcinogenic derivative of BaP. Two major groups of metabolites are formed: (1) water-soluble glutathione, glucuronide and sulfate conjugates, and (2) organo-soluble metabolites consisting of ring hydroxylated products such as phenols and dihydrodiols, quinones and labile epoxide intermediates. Figure 5-10 presents the possible metabolic pathways for BaP which are reasonably well understood.

A vital consideration in the extrapolation of risks associated with PAHs in general, and BaP specifically, is the metabolic differences, both qualitatively and quantitatively, between humans and other species. Numerous studies have demonstrated that these differences exist (Sims 1976, Wang et al. 1976, Selkirk et al. 1976). There is some evidence to indicate heritable variations in the inducibility of enzymes involved in the metabolic process occur in man but data are confounded by effects of age, seasonal variations and lack of a homogenous human population (Paigen et al. 1978). Freudenthal and coworkers (1978) reported considerable individual variations in BaP metabolism among lung microsome samples taken from the same species as well as qualitative and quantitative differences between man, monkey and rat. Similar findings have been reported by Cantrell et al. (1979) and Marshall et al.(1979). The degree of individual genetic variation of some activating enzymes is suspected to be a key factor in an organism's susceptibility to PAH-induced carcinogenesis. This issue remains unresolved at this time.

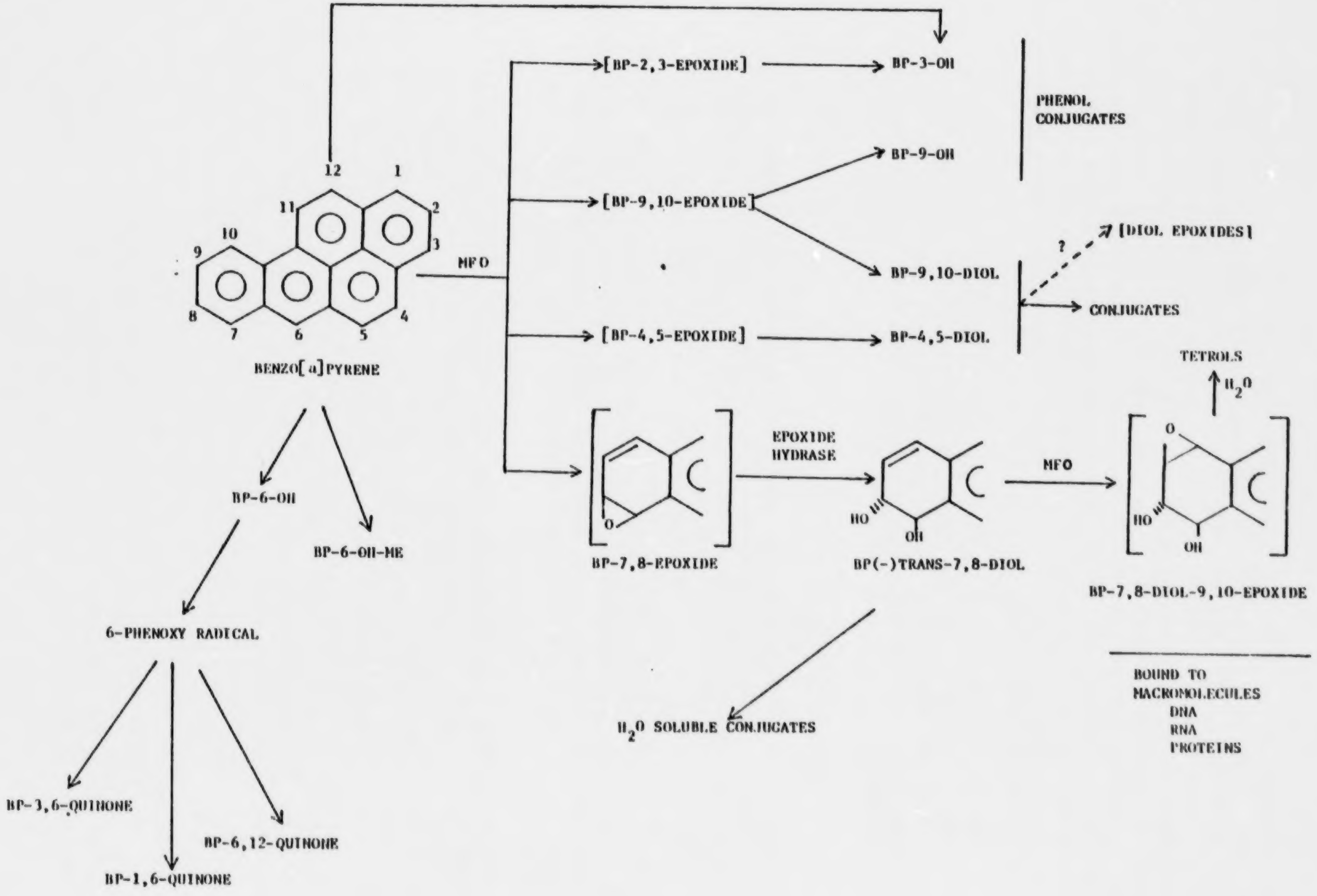
Excretion

The primary routes of BaP excretion in mice and rats are the hepatobiliary system and the feces, regardless of the route of administration. The dihydroxy-, 3-hydroxy-, and 6-hydroxy-derivatives have been detected in bile, liver and in the bowel (Berenblum and Schoenthal 1943, Falk et al. 1962, Sims 1967, 1970a,b). No extensive bioaccumulation as such is believed to occur.

5.3.1.3 Human and Animal Studies

Carcinogenicity

Several PAHs in the benzo[a]pyrene group are well established animal carcinogens, co-carcinogens and/or tumor initiators; others induce no tumorigenic responses. The capacity of individual PAHs to induce positive responses in humans is not so well established. This


12
1
11
2
10
3
9
8
7
6
5
4
BENZO[a]PYRENE
MFO
[BP-2,3-EPOXIDE]
BP-3-OH
PHENOL CONJUGATES
[BP-9,10-EPOXIDE]
BP-9-OH
BP-9,10-DIOL
?
[DIOL EPOXIDES]
CONJUGATES
[BP-4,5-EPOXIDE]
BP-4,5-DIOL
TETROLS
H_2O
O
EPOXIDE HYDRASE
MFO
HO
OH
BP-7,8-EPOXIDE
BP(-)TRANS-7,8-DIOL
BP-7,8-DIOL-9,10-EPOXIDE
BOUND TO MACROMOLECULES
DNA
RNA
PROTEINS
H_2O SOLUBLE CONJUGATES
BP-6-OH
BP-6-OH-ME
6-PHENOXY RADICAL
BP-3,6-QUINONE
BP-6,12-QUINONE
BP-1,6-QUINONE

Source: Lu *et al.* 1976; Selkirk 1977; U.S.EPA 1980.

FIGURE 5-10. POSSIBLE PATHWAYS OF BENZO[a]PYRENE METABOLISM

is primarily due to the fact that human exposures have not been to individual chemicals but rather to combinations as they occur in coke oven emissions, coal-tar, soot or from environmental exposures to tobacco smoke or exhaust fumes. Numerous studies have shown increased incidences of lung, skin and other types of cancer among workers exposed to coke oven emissions, coal gas, coal tar and pitch (IARC 1972). These studies, however, do not allow identification of the individual chemical(s) responsible, do not account for possible synergistic or co-carcinogenic effects resulting from other components, often are unable to clearly define exposure levels and generally are not amendable to quantifying human risk.

For the most part, available data for specific PAH compounds are from skin painting and subcutaneous or intramuscular injection experiments in mice. Few oral or inhalation experiments have been conducted, and those that are available are generally inadequate for risk assessment purposes.

Benzo[a]pyrene

Among the compounds in the benzo[a]pyrene group, BaP itself has been the most extensively studied. The published literature on BaP is vast. A few representative studies of the positive carcinogenic effects of BaP in a variety of animal models are presented in Table 5-47. BaP has been shown to be both a local and systemic carcinogen by oral, dermal and intratracheal routes. It is also a transplacental carcinogen, an initiator of skin carcinogenesis in mice and is carcinogenic in single dose experiments. Additional documentation of the carcinogenicity of BaP may be found in IARC (1972) or the Survey of Compounds . . . Tested for Carcinogenic Activity (1961, 1968, 1970, 1972, 1978).

Few studies have adequately examined the carcinogenic effects of orally administered BaP. Available studies are generally of very short duration with small test populations and frequently without appropriate control animals. Incidence data for the two best available oral studies for BaP (Neal and Rigdon 1967, Fedorenko and Yanysheva 1966) are presented in Table 5-48. Forestomach tumors were found in CFW mice fed BaP in their diet for an average of 110 days (Neal and Rigdon 1967) as well as in CC57 mice administered BaP by gavage once a week for 10 weeks and then maintained up to 19 months (Federenko and Yanysheva 1966). Although both these studies are by the oral route, several shortcomings raise questions as to their relevance to possible induction of cancer in humans from ingestion of BaP. Test populations in both studies were exposed for periods considerably less than lifetime and tumors were limited to the forestomach. The site of tumor development and the probable underlying mechanism are possibly unique to laboratory rodents. Unlike the fundus portion of the human stomach (which is homologous to the forestomach of rodents), the rodent forestomach is nonglandular and similar in histology to skin epithelium.

TABLE 5-47. CARCINOGENIC ACTIVITY OF BENZO[a]PYRENE BY VARIOUS EXPOSURES

Species (No.)	Major Effects	Dose	Exposure (# administrations)	Route	Incidence	Comments
A/HeJ female mice (12)	Forestomach tumors	1 mg/g diet Ave. 4.8 mg BaP/mouse exposure	3 x/wk for 2 wks	Diet	12/12	Duration 29 wks
		Controls			0/12	
Ha/ICR female mice	Forestomach tumors	1 mg/mouse in corn oil	2 x/wk for 4 wks	Gavage	36/39	Duration 21 wks
		0.3 mg/mouse in corn oil	3 x/wk for 8 wks	Gavage	33/33	Duration 31 wks
		Control (no treatment)			0/19	Duration 31 wks
Mice	Tumors	0.2 mg	Single administration	Gavage	5/11	
		0.05 mg	" "		0/9	
		0.012 mg in PEG	" "		2/10	
Sprague Dawley female rats (9)	Mammary tumors	100 mg	Single administration	Oral	8/9	
C57Bl/6J female mice (30)	Skin tumors	0.02 μmoles	Once every two wks for 60 wks	Skin painting	12/30	Mostly squamous cell carcinomas
		0.1 μmoles	(Same as above)	" "	26/27	

TABLE 5-47. CARCINOGENIC ACTIVITY OF BENZO[a]PYRENE BY VARIOUS EXPOSURES (continued)

Species (No.)	Major Effects	Dose	Exposure (# administrations)	Route	Incidence	Comments
CD-1 female mice (30)	Papillomas	200 nmoles	Single application followed by twice weekly applications of 10 μg TPA for 23 weeks	Skin painting	94%; 4.8 papillomas/ mouse	Initiator of skin carcinogenesis
Sprague Dawley rats (13)	Sarcomas	0.2 μmoles	Alternate days for 30 doses	Subcutaneous injection	13/13	Average latency 101 days
Mice, male (14)	Sarcomas	2.4 μmoles	At monthly intervals for 3 treatments	Injection	13/14	Average latency: 129 days
Mice, female (16)	"	" "	(Same as above)	"	8/16	160 days
Rats	Lung tumors	2.5 mg	Once monthly for 10 mo.	Intratracheal instillation	80%	Observed for 2 yr beyond treatment
		0.25			43%	
		0.05			28%	
		0.01			14%	
		0.002			0%	
		0.0005			0%	
		Mixed with a blood substitute BK-8 and India ink				
Syrian Golden hamsters	Respiratory tract tumors	1 mg	Once weekly for 52 weeks	Intratracheal instillation	26/28	
		0.5			25/29	
		0.25			9/30	
		0.125			4/30	
		0.0625			3/30	
		in saline				

TABLE 5-47. CARCINOGENIC ACTIVITY OF BENZO[a]PYRENE BY VARIOUS EXPOSURES (continued)

Species (No.)	Major Effects	Dose	Exposure (# administrations)	Route	Incidence	Comments
ST/A mice	Mammary tumors	10 mg	Single injection	Intraperitoneal	2/10	At one year
Pregnant ICR/Ha mice	Lung adenomas; initiation of skin carcinogenesis	2-4 mg	Dams treated on days 11, 13 and 15 of gestation	Intraperitoneal or subcutaneous injections	- - -	Transplacental carcinogen

Source of data: USEPA 1980, IARC 1972.

TABLE 5-48. INCIDENCE OF ORALLY ADMINISTERED, BENZO[a]PYRENE-INDUCED FORESTOMACH TUMORS IN MICE

Strain	Treatment	Incidence of Forestomach Tumors	Comments	Reference
CC57 mice 2-3 mo.	1 mg BaP in 0.2 ml triethylene glycol, 1x/wk for 10 wk by intubation (fasted)	23/27 (85%)	All mice were held for 19 months prior to sacrifice.	Fedorenko and Yanysheva (1966).
	As above except 0.1 mg	23/30 (77%)		
	As above except 0.01 mg	5/24 (21%)		
	As above except 1 μg	2/26 (8%)		
	As above except 0.1 μg	0/16 (0%)		
	Control	not given		
CFW mice	32.5 mg BaP mg/ kg/day in the diet	66/73 (90%)	The various treatment groups were put on test at different times. The mice at different exposure levels varied in age from 17 to 116 days at the start of the experiment and were exposed for an average of 110 days (range: 70-197 days) over a 183 day average study duration (range: 85-219 days).	Neal and Rigdon (1967).
	As above except 13 mg	19/23 (83%)		
	As above except 6.5 mg	24/34 (71%)		
	As above except 5.85 mg	4/40 (10%)		
	As above except 5.2 mg	1/40 (3%)		
	As above except 3.9 mg	0/37 (0%)		
	As above except 2.6 mg	1/23 (4%)		
	As above except 1.3 mg	0/24 (0%)		
	As above except 0.13 mg	0/25 (0%)		
	Control	0/289 (0%)		

This suggests that the rodent forestomach may be unusually sensitive to repeated localized exposure to BaP akin to skin painting bioassays. This presumably local effect of ingested BaP on rodent forestomach epithelium, in the absence of tumors at other sites, appears to be of questionable relevance to humans with a wholly different stomach anatomy.

Other PAHs in the Benzo[a]pyrene Group

The carcinogenic activity of other PAHs included in the benzo[a]pyrene group is summarized in Table 5-49. No carcinogenicity data were found for acenaphthylene; the majority of other compounds in this group have been tested only in the mouse with exposures generally limited to skin painting experiments. Only benz[a]anthracene and dibenz[a,h]anthracene have been tested by the oral route. Both were found to be carcinogenic by the oral route (IARC 1972). Dibenz[a,h]anthracene induced forestomach tumors in mice following oral administration of this compound (IARC 1972, USEPA 1980). Repeated gavage administration of benz[a]anthracene (15 doses of 1.5 mg over 5 week period) produced a 95% incidence of lung adenomas, 64% incidence of hepatomas and a 3% incidence of forestomach papillomas in the test population of 59 B6AF1/J mice. Another group of 20 mice given only two treatments 3 days apart developed 16 hepatomas and 17 lung adenomas. Controls for these test groups had 2 hepatomas, 10 lung adenomas and no forestomach tumors among 59 control animals (IARC 1972).

Dibenz[a,h]anthracene, benz[a]anthracene, benzo[b]fluoranthene, chrysene and indeno[1,2,3-c,d]pyrene are complete carcinogens for mouse skin and all are also initiators of skin carcinogenesis in this species (USEPA 1980, IARC 1972, Habs *et al*. 1980). No carcinogenic response was noted in a skin painting study with benzo[k]fluoranthene in NMRI mice treated twice a week with up to 9.2 pg/mouse/application for their lifetime (Habs *et al*. 1980).

A pronounced co-carcinogenic effect was observed in a single experiment conducted with benzo[g,h,i]perylene (2000 µg) plus 5 µg BaP applied to skin of ICR/Ha Swiss mice 3 times per week for 52 weeks (USEPA 1980).

Local sarcomas have also been produced at the site of subcutaneous or intramuscular injections of benzo[b]fluoranthene, chrysene, dibenz[a,h]anthracene and indeno[1,2,3-c,d]pyrene (USEPA 1980, IARC 1972).

Ultimate Carcinogenic Metabolites

Investigators have long sought a common molecular feature among carcinogenic PAHs such as BaP. Recent attention has focused on the increased reactivity of diol epoxide metabolites of PAHs in which the oxirane oxygen forms part of a "bay region." Carcinogenicity studies conducted with various potential oxidative metabolites of BaP, for example, suggest that the 7,8-diol-9,10-epoxide is the most active metabolite (Wislocki *et al*. 1977).

TABLE 5-49. SUMMARY OF THE CARCINOGENIC ACTIVITY FOR OTHER POLYCYCLIC AROMATIC HYDROCARBONS IN THE BENZO[a]PYRENE GROUP[1]

Chemical	Species (No.)	Major effects	Dose	Exposure (# administrations)	Route	Incidence	Comments
Benz[a]Anthracene	Mice (13)	No tumors in 16 months	0.5 mg in mineral oil	Single dose	Gavage	------	
Benz[a]Anthracene	Mice (27)	Papilloma of fore-stomach	0.5 mg in mineral oil	8 or 16 X at 3-7 day intervals	Gavage	2/27	
	Mice (16)	No tumors	Mineral oil	Vehicle controls	Gavage	0/16	
Benz[a]Anthracene	B6AF1/J mice (59)	Lung adenomas; hepatomas; papillomas (stomach)	1.5 mg/mouse methocelaerosol OF	15 treatments in 5 weeks	Gavage	56/59; 38/59; 2/59	2 hepatomas in 20 animals with median age of death 547-600 days
Benz[a]Anthracene	Mice (20)	Hepatomas; lung adenomas	1.5 mg/mouse	2 treatments, 3 days apart	Gavage	16/20; 17/20	Median death age 547-600 days
Controls	Mice (59)	Hepatomas; lung adenomas	Untreated			2/59; 10/59	
Benz[a]Anthracene	Mice (20)	Papilloma	0.4% in mineral oil	2 X/wk, 68 wks	Skin Painting	1/20	No control data
Benz[a]Anthracene	C3H/He Mice	Tumors (type not specified - mostly malignant)	.002% in toluene	3 X/wk/50 wks	Skin Painting	0/32	
			.02% " "	" " " " "	" "	1/18	
			.2% " "	" " " " "	" "	3/32	
			1.0% " "	" " " " "	" "	8/29	
			.0002% in dodecane	" " " " "	" "	4/31	Dodecane is a cocarcinogen
			.002% " "	" " " " "	" "	8/21	
			.02% " "	" " " " "	" "	4/20	
			0.2% " "	" " " " "	" "	11/21	
			1% " "	" " " " "	" "	17/22	
Benz[a]Anthracene	Mice (75)	Tumors Papillomas	.05% in acetone +5% croton oil	Alternate once weekly applications	Skin Painting	17/17 at 9 months 18/9 at 12 months	
		Tumors	controls - 5% croton oil	" "	" "	1/13	

TABLE 5-49. SUMMARY OF THE CARCINOGENIC ACTIVITY FOR OTHER POLYCYCLIC AROMATIC HYDROCARBONS IN THE BENZO[a]PYRENE GROUP[1] (continued)

Chemical	Species (No.)	Major effects	Dose	Exposure (# administrations)	Route	Incidence	Comments
Benz[a]Anthracene	Mice (20)	Skin tumors	6 mg/mouse total followed by croton oil	10 X over 5 wks weekly applications	Skin Painting	21 tumors in 7/18 mice	BA alone gave no tumors
Benz[a]Anthracene	Hamsters (10) (Syrian gold.)	No tumors	0.5% in mineral oil	Bi-weekly for 10 wks	Skin Painting	----	6/10 alive at 50 wks, all dead at 85 wks
Benz[a]Anthracene	C57BL mice	Tumors	0.05 mg in tricaprylin	Single	Injection	5/43 mice	Surviving at 9 months
			0.2 mg " "	"	"	11/43	
			1.0 mg " "	"	"	15/31	
			5.0 mg " "	"	"	49/145	
			10.0 mg " "	"	"	5/16	
Benz[a]Anthracene	C57BL mice	Sarcomas	1 mg/mouse in arachis oil	10 weekly injections	Subcutaneous	14/20 in 146-179 days	
Benz[a]Anthracene	Rats (20)	No tumors	1.9 mg in tricaprylin	Single application	Subcutaneous	0/9 alive at 14.5 months	
Benz[a]Anthracene	Rats (29 ♀)	Mammary tumors	13 mg/kg	3 injections at 3 day intervals	Injection	0/29 at 98 days	
Benz[a]Anthracene	Mice (52)	Carcinomas (bladder)	2 mg implant in paraffin wax	Single	Bladder implant.	17/52	In 40 weeks or more; 3.8% incidence in controls
Benz[a]Anthracene	BALB/C, newborn and 2-8 days old	Pulmonary adenomas, adenocarcinomas	50 µg in gelatin (aq)	Single injection	Injection	?	More tumors in newborns
Benzo[b]Fluoranthene	Mice (20/group)	Papillomas	0.5% in acetone	3 X/week	Skin painting	100% w/in 8 mos.	No Control Group
		Carcinomas	" " "	" " "	" "	90%	
		Papillomas	0.1% in acetone	" " "	" "	65% w/in 12 mos.	
		Carcinomas	" " "	" " "	" "	85%	
		Papillomas	0.01% in acetone	" " "	" "	1/10 survivors after 14 mos.	
		Carcinomas	" " "	" " "	" "	----	

TABLE 5-49. SUMMARY OF THE CARCINOGENIC ACTIVITY FOR OTHER POLYCYCLIC AROMATIC HYDROCARBONS IN THE BENZO[a]PYRENE GROUP[1] (continued)

Chemical	Species (No.)	Major effects	Dose	Exposure (# administrations)	Route	Incidence	Comments
Benzo[b]Fluoranthene	Mice (20)	No tumors	1.0 mg in acetone	Single application	Skin painting	----	63 weeks observ.
	Mice	Papillomas Carcinomas	1.0 mg in acetone and croton resin	Single application Repeated application	Skin painting " "	18/20 5/20	Initiator of skin carcinog.
Benzo[b]Fluoranthene	16 ♂, 14 ♀ XVIInc/z mice	Sarcoma at injection site	0.6 mg/injection	3 injections in 2 months	Subcutaneous	18/24	Avg. latent period of 4.5 months
Benzo[b]Fluoranthene	NMRI (40)	Papillomas, sarcomas, and carcinomas	3.4 pg in acetone 5.6 pg " " 9.2 pg " "	2 X/wk for lifetime " " " " " " " " " "	Skin painting " " " "	5.3% 14.7% 54.1%	No tumors in controls
Benzo[k]Fluoranthene	NMRI (40)	No tumors	3.4 pg in acetone 5.6 pg " " 9.2 pg " "	2 X/wk for lifetime " " " " " " " " " "	Skin painting " " " "	- - -	No tumors in controls
Chrysene	Mice (20)	Papillomas Carcinoma	1.0% in acetone	3 X/wk for ?	Skin painting	9 After 8 8 mos.	Obvious shortened life-span
Chrysene	CFI mice	Epithelioma	0.2% in acetone	Biweekly for 31wks	Skin painting	1/16	
Chrysene	Mice	No tumors	1 mg in acetone	Single application	Skin painting	-----	63 wks observ.
	Mice	Papillomas Carcinomas	1 mg in acetone and croton resin	Single application Repeated application	Skin painting " "	16/20 2/20	Initiator of skin carcinog.
Chrysene	Mice (50)	No tumors	2 mg (purified)	Single injection	Subcutaneous	-----	45 wks observ.
Chrysene	Mice (30)	Negative	10 mg/implantation	2 implantations	Subcutaneous	-----	
Chrysene	Mice (40-50) (C57BL)	Sarcomas	5 mg in trycaprylene	Single injection	Subcutaneous	5/22?	Avg. induction time = 271 days 22 alive at 150 days

TABLE 5-49. SUMMARY OF THE CARCINOGENIC ACTIVITY FOR OTHER POLYCYCLIC AROMATIC HYDROCARBONS IN THE BENZO[a]PYRENE GROUP[1] (continued)

Chemical	Species (No.)	Major effects	Dose	Exposure (# administrations)	Route	Incidence	Comments
Chrysene	Mice (57BL)	Tumors (?)	1 mg in arachis oil	10 injections	Injection	2/20	No tumors in vehicle control
Chrysene	Rats	Sarcomas	2-3 mg/injection	Repeated (?)	Injection	4/10	2/10 sarcomas in vehicle controls
Chrysene	Rats (10)	No tumors	0.05% (aq.)	Bi-weekly	Injection	0/4 at 18 mos.	
Chrysene	Mice (50)	No tumors	2 mg in lard	Single	Injection	-----	50 wk observ.
Dibenz[a,h]Anthracene	Mice	Tumors (1 carcinoma of forestomach)	9-19 mg total dose	Over 5-7 months	Dietary	7/22 after 1 year	
Dibenz[a,h]Anthracene	Mice (20)	Squamous-cell carcinoma, Papilloma of forestomach	0.4 mg/day in oil emulsion	W/in 406 days	Drinking water	2/20 11/20	
	Mice	Lung, heart and intestinal tumor	0.4 mg/day in NaOH	?	Drinking water	?	
Dibenz[a,h]Anthracene	Mice (42) DBA/2	Pulmonary adenomatosis	0.76-0.85 mg/day in olive oil	200 days	Drinking water	27/27	
		Aveologenic carcinoma	" "	" "	" "	24/27	
		Hemangio-endothelioma	" "	" "	" "	16/27	
		Mammary carcinoma	" "	" "	" "	12/13♀	
	Mice (35)	Mammary carcinoma	------------------	C O N T R O L S	------------------	0	
		Pulmonary adenomatoses	------------------	"	------------------	0	
Dibenz[a,h]Anthracene	Mice (BALB/c)	Mammary carcinoma	0.5% in almond oil (15 mg total dose)	Twice weekly for 15 weeks	Stomach tube	1/20	
	Pseudopreg. Mice (BALB/c)	Mammary carcinoma	" " " "	" " " " "	" "	13/24	

TABLE 5-49. SUMMARY OF THE CARCINOGENIC ACTIVITY FOR OTHER POLYCYCLIC AROMATIC HYDROCARBONS IN THE BENZO[a]PYRENE GROUP^1 (continued)

Chemical	Species (No.)	Major effects	Dose	Exposure (# administrations)	Route	Incidence	Comments
Dibenz[a,h]Anthracene	C3H mice	Mammary tumors " "	0.25% in benzene Controls	Twice weekly Untreated	Skin painting " "	10/11 50%	
	Swiss mice (20)	Skin tumors	30 µg/dose in acetone-benzene	Bi-weekly	Skin painting	16/20	
Dibenz[a,h]Anthracene	ICR/Ha Swiss mice	Tumors (some carcinomas)	0.001% 0.01% 0.1%	3 X weekly " " " " " "	Skin painting " " " "	1/30 43/50 (39 carcinomas) 39/40 (32 carcinomas)	
Dibenz[a,h]Anthracene	C3H mice	Local sarcomas	0.00019 mg/dose 0.0078 mg/dose 0.016 mg/dose 0.03 mg/dose 0.06 mg/dose 0.12 mg/dose 0.25 mg/dose	Single dose	Injection	2/79 6/40 6/19 16/21 20/20 21/23 19/21	Lowest effective dose - 0.0019 mg Avg. latent period = 3.7 mos.
Dibenz[a,h]Anthracene	Mice (20)	Sarcomas	0.0125 mg in lard	5 injections	Injection	4/20	
Dibenz[a,h]Anthracene	Mice (newborn)	Local sarcomas Lung Adenomas	≥ 0.08 µg ≥ 0.2 µg	5 injections	Subcutaneous Subcutaneous		Dose-related appearance increased incidence
Dibenz[a,h]An-	Rats	Tumors	8 mg/injection	9 injections at monthly intervals	Injection	11/40	W/in 1 year
Dibenz[a,h]Anthracene	Rats	Sarcomas	0.1 mg in olive oil 1.0 mg in olive oil	Single dose " "	Injection "	3/9 6/10	
Dibenz[a,h]Anthracene	Guinea pigs	Sarcomas	8-48 mg (total dose) in oil	Multiple	Subcutaneous	2/25	After 19 mos.
Dibenz[a,h]Anthracene	Fowl	Sarcomas	0.4% in lard	Single dose	i.m.	15/31	W/in 45 mos.

TABLE 5-49. SUMMARY OF THE CARCINOGENIC ACTIVITY FOR OTHER POLYCYCLIC AROMATIC HYDROCARBONS IN THE BENZO[a]PYRENE GROUP[1] (continued)

Chemical	Species (No.)	Major Effects	Dose	Exposure (# administrations)	Route	Incidence	Comments
Dibenz[a,h]Anthracene	Mice	Lung adenomas	$\geq$ 0.1 mg colloidal dispersion (aq.)	Single dose	i.v.		Dose-response relationship
Dibenz[a,h]Anthracene	Frogs	Renal adenocarcinomas	0.3-0.5 mg in olive oil	Single dose	Intrarenal	26%	3% incidence controls
Dibenz[a,h]Anthracene	Mice	Lung Tumors	36 μmoles/kg	Single dose	i.v.	10/10	
Dibenz[a,h]Anthracene	Mice	Papilloma Epithelioma	?	?	Skin painting	8/65 33/65	63% tumor incidence/239 day avg. latent period = 26 Iball index
Dibenz[a,h]Anthracene	Mice (14♀) Mice (13♂)	Aveologenic carcinoma and pulmonary adenomatosis	179 mg (total) 236 mg (total) (in olive oil)	237-279 days " " "	Drinking water	77% 100%	
Indeno[1,2,3-c,d]Pyrene	Mice (♀)	No tumors	0.05 or 0.1% in dioxane	3X/wk for 12 months	Skin painting	- - -	
Indeno[1,2,3-c,d]Pyrene	Mice	No tumors	0.01% in acetone	3 X/wk for 12 months	Skin painting	0	Dose-response Relationship
		No tumors	0.05% " "	" " " " " "	" "	0	
		Papillomas	0.1% " "	" " " " " "	" "	6/20	" " "
		Carcinomas	0.1% " "	" " " " " "	" "	3/20	" " "
		Papillomas	0.5% " "	" " " " " "	" "	7/20	" " "
		Carcinomas	0.5% " "	" " " " " "	" "	5/20	" " "
		Skin carcinogenicity	250 μg total	10 paintings at 2 day interval	" "		
Indeno[1,2,3-c,d]Pyrene	Mice ♂ Mice ♀	Sarcomas Sarcomas	0.6 mg in olive oil	3 injections at 1 month intervals	Injection	10/14 1/14	W/in 265 days W/in 145 days
Indeno[1,2,3-c,d]Pyrene	NMRI mice (40)	No tumors	3.4 pg in acetone 5.6 pg " " 9.2 pg " "	2 X/wk for lifetime " " " " " " " " " "	Skin painting " " " "	- - -	No tumors in controls.

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[1] Source of data: USEPA 1980, IARC 1972, Habs et al. 1980.

Wislocki and coworkers (1977) found that BP7,8-oxide was the most active carcinogen among 4 BaP arene oxides tested (and only slightly less effective than BaP) following skin application of 0.3-0.4 µmol to C57BL/6J mice once every two weeks for 60 weeks. Since 4-,5-,6-,7-, 8-,9- and 10-hydroxyBP were all inactive as complete carcinogens at 0.4 µmol/dose (Kapitulnik et al. 1976), carcinogenic activity could not be attributed to phenolic isomerization products of BP7,8-oxide. Further tests with metabolic products of BP7,8-oxide implicated BP7,8-dihydrodiol as a more potent proximate carcinogen; the dihydrodiol was presumably formed when BP7,8-oxide was hydrated by epoxide hydrase (Jerina et al. 1977a,b). BP7,8-dihydrodiol induced 100% incidence of skin tumors (as did BaP itself) compared to <20% incidence induced by BP7,8-oxide at doses of 0.15 µmol by the above schedule; at lower doses the dihydrodiol was more active than BaP (Levin et al. 1977a,b). The lack of carcinogenic activity of 7,8,9,10-tetrahydro-BP7,8-epoxide and 7,8,9,10-tetrahydro-BP7,8-diol at 0.3 µmol/dose indicated that the 9,10-double bond was important in metabolic transformation to ultimate carcinogenic products (Levin et al. 1976a,b, Jerina et al. 1978). The most probable candidates for this ultimate metabolite, would again be the BP7,8-diol-9,10-epoxides formed by oxidation of the 7,8-dihydrodiol at the 9,10-position by the monooxygenase system (Conney et al. 1980, Jerina et al. 1977a,b). In tests with newborn Swiss-Webster mice, intraperitoneal injection of a 28 nmol dose of either BaP, BP7,8-dihydrodiol or BP7,8-diol-9,10-epoxide-2 resulted in a definite sequential increase in tumorigenic activity of BaP, BP7,8-dihydrodiol and BP7,8-diol-9,10-epoxide-2, respectively, clearly implicating the dihydrodiol as the proximate carcinogenic metabolite and the epoxide as the ultimate carcinogenic metabolite. The BP7,8-diol-9,10-epoxide-1 isomer was too toxic for an adequate assessment of tumorigenic activity (Kapitulnik et al. 1977).

These and other studies have generated the "bay-region" (i.e., hindered area between the 10- and 11-positions) theory to correlate structure of polycyclic hydrocarbons with carcinogenic activity. Presumably benzylic carbonium ions derived from epoxides of a saturated angular benzo ring have a greater ease of formation when the carbonium ion is located in the bay region, thus enhancing the chemical reactivity and perhaps the mutagenicity and carcinogenicity of the epoxide (Jerina et al. 1977a, 1977b, 1978, Conney et al. 1980).

A number of recent experiments with bay region diol epoxides of benz[a]anthracene, dibenz[a,h]anthracene and chrysene indicate that they, also, are more active than the parent compounds or other oxidative metabolites.

Skin painting studies with various metabolites in CD-1 mice suggest that BA3,4-dihydrodiol is at least 10 times more tumorigenic than benz[a]anthracene itself or other metabolites (Wood et al. 1977b,c, Levin et al. 1978, Slaga et al. 1978). In newborn mice treated with various metabolites of benz[a]anthracene, BA3,4-dihydrodiol induced at least 30-fold more pulmonary adenomas than the parent compound or 4 other dihydrodiols tested (Wislocki et al. 1978).

Similarly, Buening and coworkers (1979b) found DBA 3,4-dihydrodiol to be equal to dibenz[a,h]anthracene and considerably greater than DBA 1,2-dihydrodiol, DBA 5,6-dihydrodiol and DBA H_4-3,4-diol in tumor-inducing activity on mouse skin following single applications. In the same study, the incidence of pulmonary adenomas induced in newborn Swiss-Webster mice following intraperitoneal injection was similar for dibenz[a,h]anthracene and DBA 3,4-dihydrodiol, while tumor incidences caused by DBA 1,2-dihydrodiol and 5,6-dihydrodiol were significantly lower. Again, the much lower tumorigenic activity on mouse skin of DBA 3,4-diol compared to DBA 3,4-dihydrodiol is consistent with the prediction of the bay-region theory that metabolism at the 1,2 double bond would yield the ultimate carcinogenic diol epoxide, DBA 3,-4-diol-1,2-epoxide (Buening et al. 1979b).

Initiation-promotion studies in CD-1 mice with chrysene and various chrysene metabolites applied once at doses of 0.4, 1.25 or 4.0 μmoles and followed by twice weekly applications with TPA, a phorbol ester, for 25 weeks resulted in significantly greater tumorigenic activity for chrysene 1,2-dihydrodiol above chrysene itself (Levin et al. 1978). Similar findings were reported by Buening et al. (1979a) who found enhanced lung tumor activity for chrysene 1,2-dihydrodiol in newborn mice given intraperitoneal injections on days 1,8 and 15 of life.

In summation, benzo[a]pyrene has been most extensively studied of all PAHs in the benzo[a]pyrene group. BaP has been shown to be both a local and systemic carcinogen by oral, dermal and intratracheal routes. It is also a transplacental carcinogen and an initiator of skin carcinogenesis in mice. Few studies have adequately examined the carcinogenic effects of orally administered BaP but it has been shown to induce forestomach tumors in mice exposed to BaP by the oral route. The absence of tumors at sites other than in the forestomach and the unique anatomy of rodent forestomach in contrast to humans raises questions as to the significance of the induction of forestomach tumors in mice to human risks associated with the ingestion of BaP.

With respect to other PAHs included in this group, benz[a]anthracene and dibenz[a,h]anthracene are both carcinogenic in mice by the oral route as well as complete carcinogens for mouse skin as are benzo[b]fluoranthene and indeno[1,2,3-c,d]pyrene. Benzo[g,h,i]perylene is a co-carcinogen with BaP and benz[a]anthracene, benzo[b]fluoranthene, chrysene and indeno[1,2,3-c,d]pyrene are all initiators of skin carcinogenesis in mice.

Recent studies on the mechanisms of carcinogenic activity of some PAHs suggest increased reactivity of the diol epoxides of PAHs in which the oxirane oxygen is located in the "bay-region." Experiments with possible metabolites of BaP indicate that one or more of the BP7,8-diol-9,10-epoxides are the ultimate carcinogenic form. The bay-region theory of polycyclic hydrocarbon carcinogenesis is further supported by findings of increased carcinogenic activity of the bay-region diol epoxides of benz[a]anthracene, dibenz[a,h]anthracene and chrysene above that of the parent compounds.

Adverse Reproductive Effects

BaP appears to exert little effect on the developing embryo (Bulay and Wattenberg 1970). Rigdon and Rennels (1964) found only one malformed fetus among 7 litters of rats from dams exposed to 1 mg BaP/g diet during gestation. Increased resorptions and dead fetuses were noted, however.

Juchau and coworkers (1978) reported that several human fetal tissues (i.e., liver, lung, kidney, adrenals and placenta) possess the requisite monooxygenase enzyme systems needed to bioactivate and biotransform BaP into metabolites that induce positive mutagenic effects in the Ames *Salmonella* assay.

No other data were available for compounds in this group.

Mutagenicity

A summary of the mutagenic activity exhibited by compounds comprising the benzo[a]pyrene group is presented in Table 5-50. No data were found for benzo[k]fluoranthene or indeno[1,2,3-c,d]pyrene. BaP is the most active mutagen of the group, exhibiting positive mutagenic responses in all of the test systems including induction of *in vivo* sister chromatid exchange in hamster cells (Bayer and Bauknecht 1977, Roszinsky-Köcher *et al*. 1979, Sirianni and Huang 1978) and chromosomal aberrations in both spermatogonia and bone marrow cells of hamsters *in vivo* (Basler and Rohrborn 1978; Roszinsky-Köcher *et al*. 1979). Studies conducted with various potential metabolites of BaP suggest that the BP7,8-diol-9,10-epoxide is the most potent mutagen in the presence of a metabolic activation system and may in fact be the ultimate carcinogen (Conney *et al*. 1977, Wislocki *et al*. 1976a,b, Wood *et al*. 1976, Huberman *et al*. 1976, Newbold and Brookes 1976).

Mixed responses have been found for several of the other compounds in this designated group. *In vivo* studies with benz[a]anthracene have been generally positive. Increased chromosomal aberrations were observed in mouse oocytes and hamster spermatogonia and bone marrow cells (Peter *et al*. 1979, Basler and Rohrborn 1978). Roszinsky-Köcher and coworkers (1979) noted a weak induction of sister chromatid exchange in hamster cells exposed *in vivo* to benz[a]anthracene. Both positive and negative results have been reported in host-mediated assays with *Salmonella typhimurium* TA1538 (Simmon 1979, Simmon *et al*. 1979, Rosenkranz and Poirier 1979). Benz[a]anthracene induced positive mutagenic

TABLE 5-50. SUMMARY OF MUTAGENIC ACTIVITY FOR COMPOUNDS COMPRISING THE BENZO[a]PYRENE GROUP

Compound	Chromosomal Aberrations In Vivo	Chromosomal Aberrations In Vitro	Sister Chromatid Exchange In Vivo	Host Mediated Assay	Mammalian Cells In Vitro	*Salmonella typhimurium* TA98	*Salmonella typhimurium* TA100	*Salmonella typhimurium* TA1535	*Salmonella typhimurium* TA1538	*Salmonella typhimurium* TM677	Unscheduled DNA Synthesis	References
Acenaphthylene										$+^{*}$		Kaden *et al.* 1979.
Benz[a]anthracene	+		±	±	−	$+^{*}$	$+^{*}$	$\pm^{*}$	$\pm^{*}$	$+^{*}$	+	Basler and Röhrborn 1978; Huberman 1977; Huberman and Sachs 1976; Kaden *et al.* 1979; Martin *et al.* 1978; Peter *et al.* 1979; Rosenkranz and Poirier 1979; Roszinsky-Köcher *et al.* 1979; Simmon 1979; Simmon *et al.* 1979.
Benzo[b]fluoranthene	−		±									Roszinsky-Köcher *et al.* 1979.
Benzo[g,h,i] perylene						$+^{**}$		$-^{**}$	$+^{**}$	$+^{*}$		Gibson *et al.* 1978; Kaden *et al.* 1979.
Benzo [a] pyrene	+	+	+		+	$+^{*}$	$+^{*}$		$+^{*}$	$+^{*}$	+	Basler and Röhrborn 1978; Bayer and Bauknecht 1977; Hopkin and Perry 1980; Hsu *et al.* 1979; Huberman 1977; Huberman and Sachs 1976; Kaden *et al.* 1979; Lake *et al.* 1978; Martin *et al.* 1978; Matsuoka *et al.* 1979; Mishra *et al.* 1978; Popescu *et al.* 1977; Rosenkranz and Poirier 1979; Roszinsky-Köcher *et al.* 1979; Rudiger *et al.* 1976; Salamone *et al.* 1979; Sirianni and Huang 1978.

Legend: − = no significant induced mutations; + = significant induced mutations; ± = variable or weak response.

* Tested with liver microsomal activation.

** Tested with ^{60}C gamma irradiation activation.

TABLE 5-50. SUMMARY OF MUTAGENIC ACTIVITY FOR COMPOUNDS COMPRISING THE BENZO[a]PYRENE GROUP (continued)

Compound	Chromosomal Aberrations		Sister Chromatid Exchange	Host Mediated Assay	Mammalian Cells In Vitro	Salmonella typhimurium					Unscheduled DNA Synthesis	References
	In Vivo	In Vitro	In Vivo			TA98	TA100	TA1535	TA1538	TM677		
Chrysene	±		±	-	-	-*	±*	-*	-*	+*		Basler *et al.* 1977; Basler and Röhrborn 1978; Huberman *et al.* 1972; Kaden *et al.* 1979; Kamei 1980; Poirier and de Serres 1979; Richter-Reichhelm *et al.* 1979; Rosenkranz and Poirier 1979; Roszinsky-Köcher *et al.* 1979; Salamone *et al.* 1979; Simmon *et al.* 1979; Simmon 1979.
Dibenz[a,h] anthracene	-		±		+	-**	+*	-**	+*	+	+	Gibson *et al.* 1978; Huberman 1977; Wood *et al.* 1978; Huberman and Sachs 1976; Kaden *et al.* 1979; Lake *et al.* 1978; Martin *et al.* 1978; Roszinsky-Köcher *et al.* 1979;

responses in in vitro bacterial tests with several strains of Salmonella typhimurium in the presence of liver microsomal activation (Kaden et al. 1979, Simmon 1979) and induced unscheduled DNA synthesis in Hela cells (Martin et al. 1978). No significant increase in the number of mutants was seen, however, in hamster cells exposed in culture (Huberman and Sachs 1976). Tests with possible metabolites of benz[a]anthracene indicate that the bay-region BA 3,4-diol-1,2-epoxides are the most potent mutagens and probable ultimate carcinogens for this compound (Wood et al. 1977a).

Weak induction of sister chromatid exchange but no induction of chromosomal aberrations of marrow cells was observed in hamsters exposed to dibenz[a,h]anthracene in vivo. Positive induction of unscheduled DNA synthesis was noted in both human epithelial and Hela cell cultures (Lake et al. 1978, Martin et al. 1978) and a significant number of mutations observed in V79 hamster cells exposed in culture in the presence of metabolic activation (Huberman and Sachs 1976). Mixed results have been reported in tests with various strains of Salmonella typhimurium (Kaden et al. 1979; Wood et al. 1978, Gibson et al. 1978).

In contrast to the generally positive findings for BaP, benz[a]-anthracene and dibenz[a,h]anthracene, results with chrysene have been either negative or, at most, only weakly positive. In vivo tests indicate no induction of chromosomal aberrations in hamster bone marrow cells (Basler et al. 1977, Basler and Rohrborn 1978), a weak sister chromatid exchange response in this species (Roszinsky-Köcher et al. 1979) and a slight increase in aberrations in mouse oocytes (Basler et al. 1977, Basler and Rohrborn 1978, Roszinsky-Köcher et al. 1979). Generally negative findings were noted in tests with Salmonella typhimurium (Basler et al. 1977, Simmon et al. 1979, Rosenkranz and Poirier 1979) and negative results reported for a host-mediated assay (Poirier and de Serres 1979).

Limited data are available for acenaphthylene, benzo[b]fluoranthene and benzo[g, h, i]perylene. A single experiment with acenaphthylene produced positive findings in one strain of Salmonella typhimurium TM677 in the presence of rat liver activation but only at concentrations that were toxic to the bacteria (Kaden et al. 1979). An in vivo study with benzo[b]fluoranthene resulted in a weak induction of sister chromatid exchange but no significant induction of chromosomal aberrations in hamster marrow cells (Roszinsky-Köcher et al. 1979). Benzo[g,h,i]perylene has been tested in Salmonella typhimurium, producing negative results in strain TA1535 (base-pair mutant) and positive results in strains TA1538 and TA98 (frameshift mutants) with ^{60}Co gamma irradiation activation (Gibson et al. 1978). Positive results were also noted in S. typhimurium TM677 in the presence of liver microsomal activation (Kaden et al. 1979).

Other Toxic Effects

The major focus of studies conducted with PAHs in the BaP group has been their potential for inducing carcinogenic effects. Overt signs of toxicity are generally not evident except for the carcinogenic PAHs and then only at doses sufficient to produce a high tumor incidence (NAS 1977).

Normally proliferating tissues are the selected targets for carcinogenic PAHs such as BaP. This may be attributable to specific attack on the DNA of cells in the S-phase of the mitotic cycle (Philips *et al*. 1973). Other contributing factors are alterations of enzyme activity and immunologic competence. A single carcinogenic dose of BaP was reported to produce a prolonged depression of the immune response to sheep red blood cells (Stjernsward 1966, 1969). Damage to the hematopoietic and lymphoid systems has been frequently reported in experimental animals (USEPA 1980).

Bond and coworkers (1981) have noted the development of atherosclerotic plaques in groups of eleven 4-week-old Sc strain chickens injected weekly for 20 weeks with 0.1, 1 or 10 mg/kg BaP dissolved in dimethyl sulfoxide. The specific route of exposure was unspecified. Solvent and untreated control groups were also included in the study. At 20 weeks, detectable lesions were found in 44, 45 and 75% of animals in the 0.1, 1 and 10 mg/kg BaP groups, respectively, compared to 50% of solvent controls and 22% of untreated control animals.

5.3.1.4 Human Risk Considerations -- Benzo[a]pyrene Group

Carcinogenicity of Benzo[a]pyrene

The quantitative estimate of potential carcinogenic risk to humans resulting from ingestion of benzo[a]pyrene (BaP) is calculated below. Ideally, human data relating carcinogenic response at known daily dose levels in the dose range of interest would be utilized in an appropriate mathematical model to predict risk. Usually, such ideal data are not available and extrapolations of risk must be made from either:

1) epidemiological data involving relatively high level exposures such as may occur in occupational settings, or

2) controlled experiments on laboratory animals involving very high total dosages.

In the first case, the overriding uncertainty is often in the data themselves; the exposure levels, lengths of exposure, and even response rates are usually "best estimates," and, furthermore, unknown factors (e.g., co-existent carcinogenic exposure) may bias the data. In the second case, the data are usually more accurate, but the quantitative extrapolation from animal models to humans is uncertain. At present, there is no universally accepted solution to this species to species extrapolation problem. In short, in the former case one has relevant data of questionable accuracy, whereas in the latter case, one has accurate data of questionable relevance. Further complicating the issue is the present lack of an indisputable basis for judging the relative merits of the various mathematical models relating dose and effect.

The available data concerning human and other mammalian effects have been presented above. Many experiments involving exposure of laboratory animals to BaP have been done, but surprisingly few experiments have been done in which significant populations of animals were given a wide range of doses of BaP. Two ingestion studies are considered whose data appear to lend themselves to dose/response extrapolation, one by Federenko and Yanysheva (1966) on CC57 mice, the other by Neal and Rigdon (1967) on CFW mice. Three commonly used mathematical models have been applied to each set of data to extrapolate the potential carcinogenic risk of BaP to humans. These assessments of potential human risk are subject to important qualifications:

- The experimental data chosen for extrapolation are for forestomach tumors induced either by weekly intubation (Federenko and Yanysheva 1966) or incorporation of BaP into the diet (Neal and Rigdon 1967). While apparently relevant with respect to route of administration, the site of tumor production and probable underlying mechanism are possibly unique to laboratory rodents because of the anatomy of the rodent stomach. The forestomach in both rat and mouse is non-glandular and has a stratified squamous epithelium with a cornified covering (Chiasson 1979). The fundus portion of the human stomach (homologous to the forestomach in rodents) is glandular and has a columnar epithelium. The similarities between the epithelium of rodents and that of skin suggest that the induction of forestomach tumors in mice may be analogous to the induction of skin tumors in skin-painting bioassays. This possibility calls into question the relevance of forestomach tumors in mice to the potential for human cancer from BaP ingestion. Although it is recognized that a carcinogen can cause different types of cancer in varying target organs in different species, even when the same route of exposure is used, it is considered possible that the forestomach tumors in mice induced by BaP may represent an unusually sensitive assay of the carcinogenicity of BaP by the oral route.

- The EPA guidelines call for the adjustment of dose between species to be based on body surface area not on body weight (USEPA 1979). In the case of forestomach tumor induction in rodents, neither adjustment would appear to reflect the presumably local effect of BaP on the rodent forestomach epithelium. If it were hypothesized that stomach tumors from BaP ingestion are unlikely in humans because of the differences in anatomy between rodent and human stomach, but other tumor sites were considered possible, one could reasonably adjust upwards the human equivalent daily dose to account for the wider distribution in the body and consequent dilution of BaP. Conceivably, however, stomach tumors (or other intestinal tract tumors) are possible in humans due to ingested BaP. The analysis that is carried out below adjusts dose on a body surface basis, but it is regarded as the probable worst case - tending to overstate the risk due to BaP ingestion in humans.

- Both animal studies were of less than lifetime duration, especially in the study by Neal and Rigdon (1967). Doses were administered for about half the duration of the experiment in both studies. In order to adjust animal dose schedule to an equivalent daily lifetime human dose, two assumptions were utilized as recommended by the USEPA (1979). First it was assumed that the response would be the same if the total dose was distributed evenly over the entire experimental lifetime of the animal. Second, it was assumed that a short-duration study attributable to either early mortality or early termination leads to an over-estimation of the dose required to produce the observed effect. The EPA guidelines (1979) suggest reducing the average daily dose by $(Le/L)^3$ where Le is the actual experimental lifetime and L is the theoretical lifetime of the experimental animal. In treating the Neal and Rigdon data, this adjustment leads to a marked reduction (∿1/40) in the effective daily dose level. These dose adjustments tend to ignore that detoxification processes and repair mechanisms generally reduce the effectiveness of low dose/long duration exposures.

The first set of data (Neal and Rigdon 1967) selected for extrapolation of possible risk is shown in Table 5-51 and indicates the incidence of forestomach tumors in CFW mice treated with BaP in their diet for an average of 110 days. The mice ranged in age from 17 to 116 days at the outset of the study.

To obtain a quantitative human risk, the human equivalent daily lifetime dose was computed as follows:

$$\underset{\text{(mg/day)}}{\text{human dose}} = \underset{\text{(mg/kg/day)}}{\text{animal dose}} \times \left(\frac{\text{110 days}}{\text{183 days}}\right) \times \left(\frac{\text{183 days}}{\text{90 wks} \times \text{7 d/wk}}\right)^3 \times \left(\frac{\text{human weight}}{\text{animal weight}}\right)^{2/3} \times \text{(animal kg weight)}$$

That is,

1 mg/day human dose is approximately equivalent to 13.7 mg/kg/day animal dose.

The second set of data (Federenko and Yanysheva 1966) selected for extrapolation is shown in Table 5-52, and indicates the incidence of forestomach tumors in CC57 mice treated with BaP in triethylene glycol

TABLE 5-51. CARCINOGENIC RESPONSE IN CFW MICE TREATED WITH BENZO[a]PYRENE

Mouse Dose[a] (mg/kg/day)	Equivalent Human Dose (mg/day)	Incidence of Forestomach Tumors	Percent
0.0		0/289	0
0.13	0.0095	0/25	0
1.3	0.095	0/24	0
2.6	0.190	1/23	4
3.9	0.285	0/37	0
5.2	0.380	1/40	3
5.85	0.428	4/40	10
6.5	0.476	24/34	71
13.0	0.95	19/23	83
32.5	2.38	66/73	90

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[a] Doses administered in diet for 110 days of a 183-day average study duration.

Source: Neal and Rigdon (1967).

TABLE 5-52. CARCINOGENIC RESPONSE IN CC57 MICE TREATED WITH BENZO[a]PYRENE

Mouse Dose[a] (mg/kg/day)	Equivalent Human Dose (mg/day)	Incidence of Forestomach Tumors	Percent
1.0	2.68	23/27	85
0.1	0.268	23/30	77
0.01	0.0268	5/24	21
0.001	0.00268	2/26	8
0.0001	0.000268	0/16	0

---

[a] BaP in 0.2 ml triethylene glycol was administered by intubation one time per week for ten weeks of 83-week (19 months) study duration, beginning when mice were 2-3 months old.

Source: Fedorenko and Yanysheva (1966).

solution by intubation once a week for 10 weeks. It is assumed that the control group response would be zero as in the previous study, since this was the response of the group treated with 0.1 μg, and the response of the group treated with 1 μg (8%) was also quite low. Thus, it is presumed that all forestomach tumors reported here are attributable to BaP ingestion.

To obtain the human equivalent lifetime daily dose, the following calculation was used:

$$\text{human dose (mg/day)} = \text{animal dose (mg/mouse/wk)} \times \frac{1\ \text{wk}}{7\ \text{days}} \times \frac{10\ \text{wks}}{83\ \text{wks}} \times \left(\frac{83\ \text{wks}}{90\ \text{wks}}\right)^{3} \times \left(\frac{\text{human weight}}{\text{animal weight}}\right)^{2/3}$$

That is, 1 mg/day (human dose) is approximately equivalent to .373 (mg/mouse/wk).

Estimation of Human Risk

The three dose/response models used to extrapolate human risk were the linear "one-hit" model, the multistage model and the log-probit model. The multistage model is actually a generalization of the one-hit model, in which the hazard rate function is taken to be a quadratic rather than a linear function of dose. The one-hit and multistage models assume that the excess probability (P) of a carcinogenic response to daily dose (D) is described by:

$$P = \frac{P(D) - P(0)}{1 - P(0)} = 1 - e^{-h(D)}$$

where h(D) is the "hazard rate" function, and P(D) and P(0) are the response rates at doses D and zero, respectively. For the one-hit model, the hazard rate function is simply BD, while in the multistage model, it is a quadratic function, $B_1D + B_2D^2$. The coefficients, B or B_1 and B_2, of the hazard rate functions are solved by least squares regression of the data using linearized forms of the model equations.

For the "one-hit" model: $BD = \log_e (1-P)$;
for the multistage model, $B_1D + B_2D^2 = \log_e (1-P)$.

The log-probit model assumes that human susceptibility varies with dose according to a log-normal distribution as follows:

$P = \phi (z)$ where $z = A + \log_{10}D$ and ϕ is the cumulative normal distribution function. Values of z are obtained from cumulative normal probability tables relating response rate P such that $\phi (z) = P$. Once z is determined, one can solve by linear regression for the "probit" intercept A in the linear equation:

$$z = A + \log_{10} D.$$

All of the data applicable to the models were utilized to calculate the parameters B, $B_1 + B_2$ or A. Although the EPA (USEPA 1979) has recommended that the data group which gives the largest value of B in the one-hit model should be used, this procedure was not followed here because of the relatively small number of animals per group. Table 5-53 presents the results of the mathematical calculations. Values of the parameters are given in the footnotes.

The observed and predicted responses at the equivalent human dose levels indicate the "goodness of fit". Within each data set, comparable fits are obtained by the three models. The extrapolated risk estimates based on the data of Neal and Rigdon (1967) and Fedorenko and Yanysheva (1966) are given in Tables 5-54 and 5-55, respectively. Due to different assumptions concerning the actual underlying mathematical relationship between dose and effect, the extrapolated risks are somewhat different at the relatively lower exposure levels typical of human environmental exposure. Regardless of the model, higher risk estimates (by about an order of magnitude) are predicted from the data of Fedorenko and Yanysheva (1966) due to the higher equation coefficients calculated for this study.

A possible cause for the difference in results for the two studies is thought to stem from the different modes of oral administration. In the study of Fedorenko and Yanysheva (1966), relatively higher doses were administered by intubation once a week, which probably resulted in higher exposure concentrations at the susceptible sites in the mouse forestomach. Alternatively, food may have diminished the bio-availability of BaP in the experiments of Neal and Rigdon (1967).

USEPA Risk Estimate

The U.S. EPA (1980) has established a zero ambient water concentration for the maximum protection of human health from potential carcinogenic effects of exposure to PAHs through ingestion of water and contaminated aquatic organisms. The water quality criterion is based on the assumption that each compound is as potent a carcinogen as BaP and that the carcinogenic effect of the compounds is proportional to the sum of their concentrations. Using a linear, non-threshold model, the EPA estimated, based on the findings of Neal and Rigdon (1967), that a concentration of 2.8 ng BaP/1 of surface water would result in an additional lifetime cancer risk of 10^{-6}.

TABLE 5-53. COMPARISON OF DOSE/EFFECT ANALYSES USING THREE MATHEMATICAL MODELS

Equivalent Human Dose (mg/day)	Response (%)			
	Observed	Predicted		
		One-hit	Multi-stage	$\log_{10}$/probit
Based on Neal and Rigdon (1967):				
.0095	0	0.9[a]	1.6[b]	1.4[c]
.095	0	8.8	14	11
.19	4	17	26	18
.285	0	24	36	23
.38	3	31	44	27
.428	10	34	48	29
.476	71	37	52	31
.95	83	60	74	41
2.38	90	90	91	58
Based on Federenko and Yanysheva (1966):				
2.68	85	100[d]	86[e]	92[f]
0.268	77	95	81	65
0.0268	21	26	16	27
0.00268	8	3	1.8	5.4
0.000268	0	0.3	0.2	0.5

[a] $P = 1-e^{-.97D}$; $B = 0.97$

[b] $P = 1-e^{-(1.67D - .272D^2)}$; $B_1=1.67$; $B_2= -0.272$

[c] $P = \phi(-.182 + \log_{10} D)$; $A = -.182$

[d] $P = 1-e^{-11.5D}$; $B = 11.5$

[e] $P = 1-e^{-(6.78D-2.25D^2)}$; $B_1 = 6.78$; $B_2 = -2.25$

[f] $P = \phi(0.96 + \log_{10} D)$; $A = 0.96$

TABLE 5-54. PROBABLE UPPER BOUNDS ON EXPECTED EXCESS CANCERS PER MILLION POPULATION DUE TO BENZO[a]PYRENE INGESTION

(Based on Neal and Rigdon (1967) Study)

	Exposure Level (μg/day)					
	0.001	.01	0.1	1	10	100
Linear Model	1	10	100	1,000	10,000	92,000
Log-Probit Model	10^{-3}	10^{-1}	14	750	14,600	120,000
Multi-Stage Model	1.7	17	170	1,700	16,000	151,000

TABLE 5-55. PROBABLE UPPER BOUNDS ON EXPECTED EXCESS CANCERS PER MILLION POPULATION DUE TO BENZO[a]PYRENE INGESTION

(Based on Fedorenko and Yanysheva (1966) Study)

	Exposure Level (μg/day)					
	0.001	0.01	0.1	1	10	100
Linear Model	11	110	1,100	11,000	106,000	670,000
Log-Probit Model	0.3	26	1,200	21,000	150,000	480,000
Multi-Stage Model	7	70	680	6,800	65,000	480,000

Possible Shortcoming in BaP Risk Calculations Utilizing Less Than Lifetime Exposures

The range of possible excess cancers in humans ingesting BaP determined in the preceding section is based on an assumption that the total effective dose is equal to dose rate times dose duration. Additional information presented by Neal and Rigdon (1967) allowed an examination of the validity of this assumption in extrapolating short duration animal exposures to BaP to the human situation. As the following discussion indicates, it appears that in the case of the two available BaP studies, this assumption may not be valid.

Neal and Rigdon (1967) varied both the daily dose of BaP incorporated into the diet and the duration of feeding of the BaP-containing diet. Animals were returned to a basal, untreated diet after a variable period on the BaP-containing diet with most animals killed between 140 and 200 days from the start of the BaP exposure.

Their data, presented in Table 5-56, were analyzed by multiple regression analysis using linear transformations of the log/probit and one-hit models. In the log/probit model:

$$P(z) = \phi(B_1 + B_2 \log_e D^a t),$$

where: z = the value of $B_1 + B_2 \log_e D^a t$ at the response rate $P(z)$, and

D = daily dose of BaP

t = number of days on BaP diet

B_1 and B_2 = the probit intercept and slope, respectively.

The total effective dose is shown to be directly proportional to t but a power function (D^a) of daily dose. The exponent "a" should be close to 1 if total effective dose can be approximated by D times t.

The linear transformation of this equation is $z = B_1 + (B_2 a \log_e D) + B_2 \log_e t$. The coefficients B_1, B_2a, and B_2 are obtained by least squares regression. From this analysis, "a" was determined to be 1.6 which is significantly greater than 1 ($p < .01$) suggesting that the total effective dose is not a simple product of D and t but is probably more accurately estimated by the power function, $D^{(1.6)} t$. This result means that dose rate has a stronger influence on response than dose duration.

This relationship between dose rate and dose duration can also be demonstrated in the one-hit model:

$$P = 1 - e^{-(BD^a t)}$$

TABLE 5-56. COMPARISON OF DOSE/EFFECT ANALYSES WHERE EFFECTIVE DOSE IS A POWER FUNCTION

		Response		
Daily Dose (D)	Duration (t)	Observed	Predicted(1)	(2)
0.10	110 (days)	0.017	0.10	0.07
0.16	110	0.025	0.23	0.15
0.18	110	0.10	0.27	0.19
0.20	∿110	0.71	0.31	0.22
0.4	∿110	0.83	0.61	0.55
1.0	∿110	0.90	0.90	0.98
1.0	2	0.11	0.07	0.07
1.1	4	0.10	0.19	0.15
1.0	5	0.44	0.20	0.16
0.9	7	0.30	0.23	0.19
0.40	30	0.67	0.27	0.20
12.0	1	0.50	0.79	0.91

Multiple Regression Results:

(1) log /probit model: $P = \phi(B_1 + B_2 a \log_e D + B_2 \log_e t)$, $B_1 = -1.957$;

$B_2 a = 1.113$; $B_2 = 0.692$, $a = 1.113/0.692 = 1.61$

(2) one-hit model: $P = 1 - e^{-(B D^a t)}$, $B = .035$ $a = 1.71$

Source of data for columns 1-3: Neal and Rigdon (1967).

where D, t and "a" are defined as above and "a" should again be close to one if D times t is a good approximation of total effective dose. The linear transformation is:

$$\log_e [-\log_e (1-P)] - \log_e t = \log_e B + a \log_e D.$$

Solving for the coefficients by multiple regression "a" equals 1.71 and B equals 0.035. Thus, the effective dose is more appropriately given by: $D^{(1.7)}$ t. Again, the exponent of D is significantly greater than one ($p \sim .01$).

Thus, both analyses indicate that in the case of BaP, dose rate is more important than dose duration in determining the incidence of BaP-induced forestomach tumors in mice. These results appear to contradict the validity of the assumptions inherent in the established guidelines (USEPA 1979) for computing risk to humans from ingestion of BaP, and suggest that in addition to the possible increased sensitivity of the rodent forestomach to tumor induction by BaP, extrapolation of high exposure/short duration animal studies to determine excess lifetime human risk may overestimate true risk.

Other Risk Considerations and Overview

Although the two animal studies discussed in the previous section demonstrate a carcinogenic response to BaP in mice by the ingestion route, the quantitative estimates of human risk must be treated with considerable caution since they are dependent on a number of stated assumptions that have high levels of uncertainty. Several considerations which seriously undermine the validity of quantitative human risk extrapolations for BaP have been discussed. For example, the mouse forestomach has a different anatomy than the human stomach and may be particularly sensitive to BaP. This difference in anatomy is indicative of the fundamental problem of species to species extrapolation. Slight differences in dose-effect relationship were obtained between the Neal and Rigdon (1967) and the Fedorenko and Yanysheva (1966) studies which were primarily attributed to the difference in mode of administration but which may also have been caused by differences in dosing schedules. Analysis of additional data from the Neal and Rigdon (1967) study indicates that the total effective dose is probably not a simple product of daily dose times duration but, in fact, may be a more complex power function in which the dose rate has a greater influence on response than treatment duration.

Another vital consideration in the extrapolation of carcinogenic risks associated with PAH exposure is the metabolic differences, both qualitatively and quantitatively, in the inducibility of the P-450 dependent microsomal mixed function oxidase system which is involved in the activation of tumorigenic PAHs to their ultimate carcinogenic forms. Considerable variations are known to exist among various species and significant individual variations have been documented for humans.

Among the other PAH compounds in this group, benz[a]anthracene, dibenz[a,h] anthracene, benzo[b]fluoranthene, and indeno[1,2,3-c,d] pyrene are complete carcinogens for mouse skin; benzo[g,h,i]perylene is a co-carcinogen with BaP; and benz[a]anthracene, benzo[b] fluoranthene, chrysene and indeno[1,2,3-c,d]pyrene are all initiators of skin carcinogenesis in mice. Thus, for the most part, published reports on the other PAHs in this group deal with localized tumor production and are generally not considered relevant to the assessment of systemic carcinogenicity. We have, therefore, focused our attention on the risks associated with BaP because (1) it is the dominant substance in this group with respect to human exposure (see Section 5.3.2); and (2) reduction of BaP levels will presumably lead to reduction in exposure to the other PAHs in this group. In addition to ingestion studies, BaP has been shown to be animal carcinogen by dermal and intratracheal routes. It is also a transplacental carcinogen, an initiator of skin carcinogenesis in mice, and is carcinogenic in single dose experiments.

Benzo[a]pyrene is also the most active mutagen of the group, inducing *in vivo* chromosomal aberrations in both hamster spermatogonia and bone marrow cells and inducing positive mutagenic responses in sister chromatid exchange tests. Mixed mutagenic responses have been reported for the other PAHs in this group. Because of its mutagenicity, BaP exposure could also be expected to contribute to the genetic burden of a population, but since extrapolation procedures for genetic risks have not been well established, a quantitative risk assessment for these kinds of health hazards is not presently feasible.

BaP appears to exert little effect on the developing embryo; data were unavailable for the other compounds in this group regarding adverse reproductive effects.

The major focus of studies conducted with PAHs in this group has been their potential for inducing carcinogenic effects. Thus, little information is available on other possible toxic effects associated with exposure to these compounds. However, in assessing the risks to humans associated with exposure to these PAHs, one should not overlook possible augmentation of effects through synergistic or co-carcinogenic mechanisms. Current understanding of the co-carcinogenesis process, however, is not sufficiently adequate to allow estimation of human risk at this time.

5.3.2 Human Exposure

5.3.2.1 Introduction

As is apparent from the preceding sections, monitoring data are limited for the PAHs comprising the BaP group. This section examines human exposure to benzo[a]pyrene and the other chemicals in this group via ingestion (food and drinking water), inhalation, and dermal contact.

5.3.2.2 Ingestion

Drinking Water

Basu and Saxena (1978) sampled ten finished water supplies for several PAHs, including BaP, benzo[g,h,i]perylene, indeno[1,2,3-c,d]-pyrene, and benzo[k]fluoranthene. All of these chemicals were detected in at least seven of the 10 samples (see Table 5-38). Most values range from 0.2 ng/l-1 ng/l, and these as well as maximum reported values are shown in Table 5-57. Removal efficiencies ranged from 77-100% for plants using activated carbon; thus in plants of this type, concentrations of BaP and related compounds would be expected to be low.

No data on drinking water levels were found for the other chemicals in this group (chrysene, benz[a]anthracene, acenaphthylene, benzo[b]-fluoranthene, and dibenz[a,h]anthracene). In addition, these chemicals are rarely detected in ambient water (see Section 5.2.2). Table 5-57 shows estimated exposures for these other compounds based upon the limited data available.

Food

Levels of the PAHs in raw foods appear to be generally low. However, vegetables or fruits grown in the vicinity of air releases may contain higher levels (U.S. EPA 1980). The highest levels of the BaP group PAHs in food, however, appear to result from the cooking process, especially charcoal broiling and smoking. Table 5-58 estimates daily exposure by consumption of such foods, and lists the various assumptions made in deriving the estimates. The data on contamination levels were taken from the U.S. EPA Criteria Document (U.S. EPA 1980) and White and Vanderslice (1980). It is apparent that data are limited for many of these chemicals. As a result, the columns in Table 5-58 were not totaled, since the exposure estimates shown represent only an unknown portion of the actual total exposure. In addition, the maximum intakes represent a level of maximum contamination and consumption of the food item. Hence the total is meant to represent a worst case and is not representative of widespread exposure. Charcoal broiling represents the major source of exposure to the BaP group PAHs via food. Consumption of large amounts of food cooked in this manner could result in a high exposure, perhaps up to about 6 μg/day (intake from charcoal-broiled beef and smoked pork).

TABLE 5-57. ESTIMATED HUMAN EXPOSURE TO THE BENZO[a]PYRENE GROUP PAHs VIA DRINKING WATER

	Concentration (ng/l)[a]		Estimated Daily Exposure (ng/day)[b]	
	Typical	Maximum	Typical	Maximum
Benzo[a]pyrene	0.3	1.6	0.6	3.2
Acenaphthylene	NA[c]	NA		
Benz[a]anthracene	NA	NA		
Benzo[b]fluoranthene	NA	NA		
Benzo[k]fluoranthene	0.3	0.7	0.6	1.4
Benzo[g,h,i]perylene	2.0	4.0	4.0	8.0
Chrysene	NA	NA		
Dibenz[a,h]anthracene	NA	NA		
Indeno[1,2,3-c,d]pyrene	1	1.7	2	3.4

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[a] For locations see Table 5-38. Typical concentrations are average values, the data on BaP from New Orleans was excluded from the average.

[b] Intake assumed to be 2 liters of water per day (ICRP 1975).

[c] NA = Not Available.

Source: Basa and Saxena, 1978.

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TABLE 5-58. LEVELS OF BENZO[a]PYRENE GROUP PAHs IN FOOD AND ESTIMATED EXPOSURE VIA INGESTION OF FOOD

				Benzo[a]pyrene				Benz[a]anthracene				Benzo[g,h,i]perylene				Chrysene			
		Consumption (g/day)		Contamination (µg/kg)		Intake (µg/day)		Contamination (µg/kg)		Intake (µg/day)		Contamination (µg/kg)		Intake (µg/day)		Contamination (µg/kg)		Intake (µg/day)	
		Typical	Max.	Typical	Max.	Typical	Max.	Typical	Max.	Typical	Max.	Typical	Max.	Typical	Max.	Typical	Max.	Typical	Max.
Charcoal broiled beef[a]																			
Hamburger		10	NA	NA	2.6	NA	0.03	NA	3	NA	.03	1	15	0.01	0.15	NA	1	NA	0.01
Steak		3	86	5	50	0.02	4.3	3	31	0.009	2.7	6	12	0.02	1.0	1	9	.003	0.8
Smoked pork[b]		1	27	2	55	0.002	1.5	3	33	0.003	0.9	2	25	0.002	0.7	NA	2.2	NA	0.06
Smoked sausage[c]		1.5	30	NA	4	0.006	0.12	0.2	2	0.0003	0.06	NA	3.0	0.005	0.09	NA	NA	NA	NA
Smoked fish[d]		0.1	14	1	37	0.0001	0.5	1	189	.0001	2.6	1	2.4	0.0001	0.03	1	171	0.0001	2.4
Oil		18	NA	1	8	0.02	0.14	1	30	0.02	0.5	1	4.0	0.02	0.07	NA	12	NA	0.2
Fruits		205	NA	0.02	6	0.005	1.2	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Grains		256	NA	NA	0.3	NA	0.08	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Vegetables	Total	248	NA	0.01	0.1	0.002	0.2	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Leafy	40	NA	NA	7.5	NA	0.3	NA	50	NA	NA	NA	8	NA	0.3	NA	80	NA	3.2

[a] Consumption of beef - 86 g/day, 15% charcoal-broiled - 80% hamburger, 20% steak. Worst case maximum 86 g consumption of charcoal-broiled steak.

[b] Consumption of pork - 27 g/day, 5% smoked. Worst case maximum, 27 g/day smoked.

[c] Consumption of sausage - 30 g/day, 5% smoked. Worst case maximum 30 g/day smoked.

[d] Consumption of fish - 14 g/day, 1% smoked. Worst case maximum, 14 g/day smoked.

Note: Typical consumption refers to average consumption for males 23-34; typical concentrations are not actual arithmetic averages due to the widely varied nature of the studies, but are meant to approximate such an average.

Source: USDA (1978, 1980), U.S. EPA (1980), White and Vanderslice (1980).

5.3.2.3 Inhalation

The U.S. EPA (1980) has summarized the available data for PAH levels in air, mostly for urban areas. These data, although limited, were utilized in developing exposure estimates for an assumed respiratory flow of 20 m^3/day (ICRP 1975). These results are shown in Table 5-59. As was found for food and drinking water, BaP generally appears to represent the largest exposure of chemicals in this group.

As discussed in Section 5.2.4, Moschandreas et al. (1980) examined BaP concentrations indoors. These authors found concentrations indoors to be similar to those outdoors on non-woodburning days, but higher indoor concentrations on wood-burning days. The mean level indoors on woodburning days, however, was 4.7 ng/m^3, which is within the range for urban and rural shown in Table 5-59.

In addition to ambient air, smoking can contribute to inhalation of PAHs. Schmeltz et al. (1975) reported that mainstream smoke contained 0.025 µg BaP and 0.044 µg benz[a]anthracene per cigarette. A sidestream:mainstream ratio of 3.4 for BaP was also reported, resulting in a sidestream smoke content of 0.085 µg BaP per cigarette. Thus, exposure of smokers to BaP in mainstream smoke could range from .025-2.5 µg/day, depending upon the type of cigarette smoked, the amount inhaled, and the number of cigarettes smoked, assuming a range of consumption of 1-100 cigarettes per day (U.S. DHEW 1979). An estimated 33.2% of adults over 17 years old smoke cigarettes or 54.1 million persons in the United States. Of smokers, 25-30% smoke more than 25 cigarettes per day (U.S. DHEW 1979); thus, a large segment of the population could be exposed to BaP at levels greater than 0.6 µg/day.

In addition, nonsmokers may be exposed to BaP through inhalation of sidestream smoke. Although only a few measurements of BaP have been taken in smoke-filled rooms, concentrations may be estimated from measurements of CO levels, which have been summarized by Burns (1975). The results are not consistent, and apparently depend upon a number of variables. They show levels of 44-92 mg/m^3 CO in rooms (38-92 m^3) where 30-80 cigarettes had been smoked with no ventilation. The Surgeon General's Report (U.S. DHEW 1979) reported levels up to 50 mg CO produced in sidestream smoke per cigarette, as compared with the 0.085 µg BaP produced per cigarette. Thus, by analogy, a room concentration of 0.08-0.2 µg/m^3 can be calculated. Alternatively, using 0.085 µg BaP/cigarette, and assuming a room size of 48 m^3 with no ventilation in which 40 cigarettes were smoked, a concentration of about 0.07 µg/m^3 can be calculated. A nonsmoker exposed to such a situation 2 hours/day would receive about 0.25-0.7 µg/day, assuming a respiratory flow of 1.8 m^3/hr. These values would tend to overestimate exposure since no ventilation was assumed, and a high respiratory flow was used in the calculation. Smokers in the same situation would receive the combined intake from sidestream and mainstream smoke.

TABLE 5-59. ESTIMATED EXPOSURE TO BENZO[a]PYRENE GROUP PAHs VIA INHALATION OF AMBIENT AIR

	Ambient Concentration (ng/m^3)[a]		Intake (ng/day)[b]	
	Urban	Rural	Urban	Rural
Benzo[a]pyrene	1-100	0.01-10	20-2000	0.2-200
Benz[a]anthracene	0.1-20		2-400	
Benzo[b]fluoranthene	0.1 -10		2-200	
Benzo[k]fluoranthene	0.1-10		2-200	
Benzo[g,h,i]perylene	0.2 -50	7-50[c]	4-1000	140-1000
Chrysene	0.2 -10		4-200	
Indeno[1,2,3-c,d]pyrene	0.3 -1.3		6-30	

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[a] U.S. EPA (1980), White and Vanderslice, 1980.

[b] Based on respiratory flow of 20 m^3/day (ICRP 1975).

[c] There are not enough monitoring data to explain this apparent lack of difference between urban and rural areas.

There are problems with using CO levels to estimate BaP levels in a smoke-filled room since they would be due to sidestream smoke and exhaled mainstream smoke. In addition, the concentration of CO and BaP would be influenced by type and amount of tobacco smoked, extent of inhalation, size of room, ventilation, and duration of exposure. The estimates provided above are probably on the high side since worst-case assumptions were frequently made.

The U.S. EPA (1978) estimated the sizes of populations exposed to various levels of BaP in the United States using SRI data for areas with coke ovens and ambient data or national average concentrations for other areas. Their results are shown in Table 5-60.

5.3.2.4 Dermal Contact

No direct information is available regarding the dermal exposure of humans to this group of PAHs. However, due to the low levels found in drinking water, dermal exposures are expected to be low.

5.3.2.5 Overview

Estimated typical daily exposures to the BaP group PAHs are shown in Table 5-61. Data are unavailable for estimating exposures to some of the compounds in the group. Smoking appears to be the most significant exposure route for benzo[a]pyrene and benz[a]anthracene. A smoker consuming 25 cigarettes per day could be inhaling 600 ng and 1100 ng of these compounds, respectively.

Food, primarily charcoal-broiled and smoked meats and fish, is also a significant exposure medium for BaP, benzo[g,h,i]perylene, and benz[a]anthracene, although inhalation exposures at the upper limit of the concentration ranges reported for urban areas are greater in every case. By contrast, although monitoring data are very limited, drinking water appears to contribute relatively small amounts to typical daily exposures.

Data concerning levels of these compounds in foods are variable, as are atmospheric concentrations reported for urban areas. Therefore, some subpopulations (i.e., those consuming large amounts of charcoal-broiled and smoked meat) may be exposed to considerably higher levels (possibly as high as 6 μg/day from food consumption alone).

TABLE 5-60. ESTIMATED SIZE OF THE U.S. POPULATION EXPOSED TO RANGES OF BENZO[a]PYRENE CONCENTRATIONS IN AMBIENT AIR

Population (1000's) exposed to BaP Concentration (ng/m^3)			
>5.0	1.0-5.0	0.5-1.0	<0.5
1059	102,132	26,731	73,294

NOTE: For some locations for which monitoring data were unavailable (representing about 50% of the population) the upper limits of 95% confidence intervals of national average concentrations were used. These levels were as follows:

urban SMSA	1.3 ng/m^3
urban non-SMSA	1.4 ng/m^3
rural	0.23 ng/m^3

Source: U.S. EPA (1978).

TABLE 5-61. ESTIMATED HUMAN EXPOSURE TO THE BENZO[a]PYRENE GROUP PAHs

	Typical[a] Exposure (ng/day)								
Exposure Route	BaP	Acenaphthylene	Benz[a]-anthracene	Benzo[b]-fluoranthene	Benzo[k]-fluoranthene	Benzo[g,h,i]-perylene	Chrysene	Dibenz[a,h]-anthracene	Indeno[1,2,3-c,d]-pyrene
Ingestion									
Drinking Water	0.6	0.6	NA	NA	NA	0.4	NA[b]	NA	2
Food	50	NA	30[c]	NA	NA	60[c]	3[c]	NA	NA
Inhalation									
Urban	10-60	NA	4-90	2-30	4-100	4-180	4-130	NA	6-30
Rural	2								
Smoking	600		1100						

[a] "Typical" as used here is a qualitative estimate based on average consumption and a range of reported concentrations.

[b] NA = not available.

[c] Very limited data are available concerning the dietary intake of these compounds. Thus, this only represents a partial estimate.

Source: For assumptions and references, see text.

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5.4 EFFECTS AND EXPOSURE--NON-HUMAN BIOTA

5.4.1 Effects on Non-Human Biota

5.4.1.1 Introduction

No data from acute toxicity bioassays were available for benzo[a]pyrene or any other PAHs in this group, but several studies have been done that indicate the general concentration ranges that cause toxic effects in marine and freshwater organisms.

5.4.1.2 Plants and Microorganisms

In studies on the effects of PAHs on *Escherichia coli*, BaP was found to be highly toxic when in colloidal suspension in the presence of light and oxygen. The authors suggest that the toxicity of BaP was probably due to its photo-oxidation products. The concentrations of BaP at which toxicity occurred, however, were not reported (Harrison and Raabe 1967).

Haas and Applegate (1975) studied the effect of PAHs at concentrations of 10^{-5}, 10^{-6}, and 10^{-7} M on *E. coli*; (approximate calculated concentration range, 25-2500 μg/l BaP). Benz[a]anthracene, dibenz[a,h]anthracene, and benzo[a]pyrene all promoted growth in bacteria at these concentrations.

Similar growth-stimulating effects of PAHs have been shown with freshwater algae. Graff and Nowak (1966) reported that concentrations of 10-20 μg/l of BaP, benz[a]anthracene, benzo[b]fluoranthene, indeno[1,2,3-c,d]pyrene, and benzo[g,h,i]perylene promoted growth in the algae *Chlorella vulgaris*, *Scenedismus obliquus*, and *Ankistrodesmus oraundii*.

5.4.1.3 Animals

No standard toxicity tests were found for the compounds in this group. In the only study examining mortality, benz[a]anthracene was found to cause 87% mortality to the freshwater fish, bluegill (*Lepomis macrochirus*) at 1.0 μg/l in 6 months (Brown *et al*. 1975).

Various other effects have been reported in the literature. For example, Haranghy (1956) reported that concentrations of 0.2-1.0 μg/l BaP injected into the visceral sac of freshwater mussels resulted in a significant decrease in filtration rate. The author suggested that BaP inhibited ciliary activity or possibly the entire metabolism of the mussels. Exposure of marine calcarious sponges, *Leucosolenia complicata* and *L. variabilis*, to 5 g/l BaP caused tissue damage and abnormal growth (Korotkova and Tokin 1968).

Several studies on aquatic invertebrates and lower vertebrates, including sponges, newts, and toads, have shown that PAHs in this group can produce cancer-like growths and cause teratogenetic and mutagenic effects. The number of species examined is small; however, though no exact concentration levels for these effects were given, it is believed that they were caused by concentrations higher than those generally found in the environment (Neff 1979).

Flat fish (English sole, Parophrys vetulus) exposed to hydrocarbon-contaminated sediments near the industrialized City of Seattle were found to have a significant number (32% of the adult population) of liver tumors (Malins 1979). It appeared that these fish had absorbed PAHs and PCBs from the sediment. Fish of the same species taken from relatively "clean" areas of the Washington coast were almost free of this disease. PAHs specifically were not detected in liver samples of these fish, but the authors theorized that these compounds had been absorbed to some degree and metabolized. It is known that metabolites (e.g., diol-epoxides) of certain higher-molecular-weight aromatic hydrocarbons are carcinogenic, but these compounds were not identified in liver samples in this study (Malins 1979).

In related work, Malins (1979) showed that liver enzymes of English sole extensively metabolize benzo[a]pyrene to intermediates that interact with DNA. Such interactions are thought to be a starting point for tumor formation. Studies with freshwater trout (Salmo trutta lacustris) have shown that trout liver microsomes actively biotransformed benzo[a]pyrene into a variety of electrophilic metabolites and catalyzed binding of activated BaP to DNA. Similar studies showed that liver enzymes from coho salmon (Onchorhynchus kisutch) and starry flounder (Platichthys stellatus) extensively metabolized BaP into reactive intermediates that bind to DNA (Varnasi et al. 1979). Although no data on BaP concentrations found in the coastal Washington sediments were available, it is strongly felt (Malins 1979) that the liver disease seen in the benthic English sole was associated with contaminated sediments.

5.4.1.4 Conclusions

Since the toxicity data for the BaP group PAHs are very limited, it is not possible to make any conclusions as to the toxicity of BaP or any other PAHs. The toxicity data that are available are summarized below:

Concentration (µg/1)	Compound	Effect
1.0	(benz[a]anthracene)	Caused 87% mortality in Bluegill in 6 months.
10-20	(BaP, benz[a]anthracene, benzo[b]-fluoranthene, indeno[1,2,3-c,d]-pyrene, benzo[g,h,i]perylene)	Promoted growth in freshwater algae.
25-2500	(benz[a]anthracene, dibenz[a,h]-anthracene, and BaP)	Promoted growth in bacteria.

5.4.2 Exposure of Non-human Biota

5.4.2.1 Introduction

PAHs are found in various aquatic and terrestrial environments. From monitoring data, it appears that higher concentrations are generally found near industrialized areas, major ports, and areas of petroleum or other fossil-fuel related activities. In some remote locations, such as the coast of Greenland and areas of peat deposits, concentrations of PAHs, particularly BaP, may be higher than general background levels in "pristine" areas. The source of these high concentrations is believed to be biosynthesis of PAHs from naturally-occurring quinones in anaerobic sediments, and leaching of BaP from peat deposits, which are known to contain high concentrations of PAHs (Neff 1979).

This section will examine the exposure of biota to PAHs, considering both the levels and the types of locations where it may occur.

5.4.2.2 Monitoring Data

The STORET monitoring data provide a basis for examining aquatic exposure. Summary descriptions and concentration distributions for each PAH in the BaP group are given in Section 5.2.2. Observations of values greater than the detection limit (unremarked data) are infrequent, with only 17 such observations of a total of 3,261 observations in water for this group. A greater percentage (14%) of the sediment values were unremarked; 83 of 600 total observations. Unremarked observations for both sediment and surface water are presented in Table 5-62.

Overall there were three observations of the PAHs in the BaP group greater than 1000 µg/kg in sediment.

Many of the higher sediment values occurred consistently in several locations, including Puget Sound, coast of Washington State and Oregon, San Francisco Bay and north coastal California, a hazardous waste site in North Carolina, and the Houston ship channel.

5.4.2.3 Aquatic Fate

Environmental conditions have a significant influence on the disposition of BaP and related PAHs in aquatic systems and, hence, on the exposure conditions. These compounds are quite insoluble in water and chemical transformation processes; e.g., photolysis, are not as significant as with the lower-molecular-weight PAHs. EXAMS data for BaP indicate that in five of the six environments examined, nearly all (∼98%) of the compound is accumulated in the sediments. These observations are confirmed by monitoring data that indicate a propensity of these PAHs to accumulate in the sediments. In the oligotrophic lake, a greater percentage (18.75%) of BaP remained in the water column, but

TABLE 5-62. SUMMARY OF UNREMARKED OBSERVATIONS OF CONCENTRATIONS OF BENZO[a]PYRENE GROUP PAHs IN SURFACE WATER AND SEDIMENT--STORET, 1980

Compound	Number of Observations	Range of Observations
	Surface Water (μg/l)	
Acenaphthylene	6	0.01-0.12
Benz[a]anthracene	5	1-400
Benzo[k]fluoranthene	5	320-1500
Chrysene	1	0.02
	Sediment (μg/kg dry weight)	
Benzo[a]pyrene	11	0.02-1400
Acenaphthylene	12	0.002-93
Benz[a]anthracene	12	6.2-340
Benzo[k]fluoranthene	12	0.9-1300
Benzo[g,h,i]perylene	5	4.3-40.9
Chrysene	11	0.06-120
Dibenz[a,h]anthracene	2	15.7-2600

Source: See Table 5-34.

the remainder still went to the sediments. BaP in sediment is less likely to be physically transported than BaP in the water column. It is likely to persist chemically unchanged because the conditions conducive to chemical degradation (light, oxygenation) are not present. In addition, microbial biodegradation is not a significant fate process for any of the BaP group compounds. These characteristics suggest that although monitoring data for sediment are limited, this medium may present an important exposure route. Therefore, uptake from sediment was examined.

5.4.2.4 Factors Affecting Bioavailability of Sediment Concentrations

It is known that aquatic biota are exposed to PAHs directly from water, but the extent to which they may be exposed to sediment concentrations of these compounds is less well understood.

Accumulation of hydrocarbons, including PAHs from oil-contaminated sediments by the English sole *Parophrys vetulus* has been investigated (McCain *et al*. 1978). Results from several separate studies (Roesjadi *et al*. 1978, Fucik *et al*. 1977, Anderson *et al*. 1974) indicate that overall uptake of PAHs is not significant, but is greater by suspension feeding benthic invertebrates than deposit feeders, and that accumulation by fish is greater from water than from sediment. The conclusion has been made that any PAH that is taken up comes, to a greater extent, from dissolved PAH in interstitial waters and from PAH in the water column (dissolved and/or adsorbed onto suspended solids) rather than desorbed from the sediment itself (Neff 1979).

These studies also indicated that sediment adsorbed PAHs are not readily metabolized by benthic invertebrates. However, based on the few species studied, it is impossible to quantify the degree to which accumulation of PAHs in sediment represents a major source of exposure to aquatic biota.

5.4.2.5 Conclusions

Scattered observations for the PAHs in this group reveal that they are detected infrequently in water and when found are in the low µg/l range. Unremarked observations indicate that these PAHs may be found to a greater extent and in higher concentrations in sediment. The possible contribution of naturally-occurring PAHs to these levels is unknown. Sediment concentrations do appear to be consistently higher near industrialized areas, however, and levels as high as 2600 µg/kg have been detected.

Environmental fate and monitoring data indicate that BaP group PAHs may accumulate to potentially high levels in the sediments. The extent to which these compounds are available to biota from the sediments is not well understood or quantifiable at this time, but experimental evidence indicates that although some PAHs are taken up by benthic organisms directly from sediment, the PAHs present in interstitial waters and adsorbed to suspended particulates are more available for uptake by biota.

5.5 RISK CONSIDERATIONS

5.5.1 Introduction

An objective of the risk assessment process is the quantification of risks to various subpopulation groups of humans and other classes of biota. Quantifying such risks requires:

- Careful identification of the subpopulations at risk and the populations exposed;

- Evaluation of the ranges of exposure for each subpopulation;

- Determination of the effects levels or dose-response data in the species of concern and/or proxies for these species (for example, laboratory animals as a proxy for humans); and

- Extrapolation of dose/response data to the subpopulations at risk.

The largest data base available for estimating the risks associated with human exposure to PAH compounds in the benzo[a]pyrene group is that available on BaP. Dose-response data for BaP-induced forestomach tumors in mice and information on the range and types of human exposure to BaP are sufficient to allow estimation of the potential risks associated with continuous lifetime exposure to some level of BaP. Information on exposure and effects for the other PAHs in this group is lacking and no quantitative estimates of risks associated with exposure to these compounds is possible at this time.

Extensive uncertainties are inherent in the extrapolation of carcinogenic findings in laboratory animals to humans. Additionally, because of an inadequate understanding of the mechanisms of carcinogenesis, a scientific basis has not been developed for selecting among several alternative dose-response models, which yield widely differing results. Therefore, we have applied three dose-response models to the data, and the results provide a range of potential human risk associated with exposure to BaP.

In the case of risks to other forms of biota, insufficient data are available on most toxic effects and on exposure levels to assess risk quantitatively.

5.5.2 Human Exposure

A series of possible exposure routes for humans with an indication of the size of the population at risk and typical exposure levels are presented in Table 5-61 and also in Table 5-63 with the estimated population sizes. Exposure levels of BaP appear to be the largest of the chemicals in the group via drinking water, food, and air (see Section 5.3.2), but this may be because monitoring data are limited for the other compounds. Smoking appears to be the most significant exposure route for benzo[a]-pyrene and benz[a]anthracene. A smoker consuming 25 cigarettes per day could inhale 600 ng and 1100 ng of these compounds, respectively.

Food, primarily charcoal-broiled and smoked meats and fish, is also a significant exposure medium for BaP, benzo[g,h,i]perylene, and benz[a]-anthracene, although inhalation exposures at the upper limit of the concentration ranges reported in urban settings are greater in every case. By contrast, although monitoring data are very limited, drinking water appears to contribute relatively small amounts to typical daily exposures.

Data concerning levels of these compounds in foods are variable, as are atmospheric concentrations reported for urban areas. Therefore, some subpopulations (i.e., those consuming large amounts of charcoal-broiled and smoked meat) may be exposed to considerably higher levels (possibly as high as 6 μg/day from food consumption alone).

5.5.3 Human Risk

5.5.3.1 Carcinogenicity

Several PAHs in the benzo[a]pyrene group are well established animal carcinogens, co-carcinogens and/or tumor initiators; others have not been demonstrated to induce tumorigenic responses (see Section 5.3.1.3). The capacity of individual PAHs to induce positive responses in humans is not so well established. This is primarily due to the fact that human exposures have not been to individual chemicals but rather to combinations as they occur in coke oven emissions, coal-tar, soot or from environmental exposures to tobacco smoke or exhaust fumes. Numerous studies have shown increased incidences of lung, skin and other types of cancer among workers exposed to coke oven emissions, coal gas, coal tar and pitch (IARC 1972). These studies, however, do not allow identification of the individual chemical(s) responsible, do not account for possible synergistic or co-carcinogenic effects resulting from other components, often are unable to clearly define exposure levels and generally are not amenable to quantifying human risk.

For the most part, available data for specific PAH compounds are from skin painting and subcutaneous or intramuscular injection experiments in mice. Few oral or inhalation experiments have been conducted, and those that are available are generally inadequate for risk assessment purposes.

TABLE 5-63. ESTIMATED HUMAN EXPOSURE TO THE BENZO[a]PYRENE GROUP PAHs

Exposure Route	Population[a] Size	Typical[b] Exposure (μg/day) Benzo[a] pyrene	Acenaphthylene	Benz[a]-anthracene	Benzo[b]-fluoranthene	Benzo[k]-fluoranthene	Benzo[g,h,i]-perylene	Chrysene	Dibenz[a,h]-anthracene	Indeno [1,2,3-c,d]-pyrene
Ingestion										
Drinking Water	220.6×10^6	0.0006	0.0006	NA[c]	NA	NA	0.0004	NA	NA	0.002
Food	220.6×10^6	0.05	NA	0.01[d]	NA	NA	0.06[d]	0.003[d]	NA	NA
Inhalation										
Urban	165.6×10^6	0.02 - 2	NA	0.002 - 0.4	0.002 - 0.2	0.002 - 0.2	0.004 - 1	0.004 - 0.2	NA	0.006 - 0.01
Rural	55.1×10^6	0.0002 - 0.2					0.14 - 1			
Smoking	54.1×10^6	0.6		1.1						

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[a] U.S. Department of Commerce (1980).

[b] "Typical" as used here is a qualitative estimate based on average consumption and a range of reported concentrations.

[c] NA = not available.

[d] Very limited data are available concerning the dietary intake of these compounds. Thus, this only represents a partial estimate.

SOURCE: For assumptions and references, see Section 5.3.2.

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Benzo[a]pyrene has been most extensively studied of all PAHs in the benzo[a]pyrene group. BaP has been shown to be both a local and systemic carcinogen by oral, dermal and intratracheal routes in animals. It is also a transplacental carcinogen and an initiator of skin carcinogenesis in mice. Few studies have adequately examined the carcinogenic effects of orally administered BaP but two ingestion studies whose data appear to lend themselves to dose/response extrapolation, one by Neal and Rigdon (1967) on CFW mice, the other by Fedorenko and Yanysheva (1966) on CC57 mice, resulted in the production of forestomach tumors in this species. Dose-response values based on these two studies were estimated using three extrapolation models (see Section 5.3.1.4). A summary of the risk estimates obtained for these two data sets from the various models is presented in Table 5-64. Risk estimates are shown for continuous lifetime daily exposures ranging from one nanogram to 100 micrograms. The gap between the estimates is large in the low-dose region. However, present scientific methods do not permit a more accurate or definitive assessment of human risk.

The interpretation of these calculated risk estimates is subject to a number of important qualifications and assumptions which were discussed in Sections 5.3.1.3 and 5.3.1.4:

- The site of tumor production and probable underlying mechanisms are possibly unique to laboratory rodents. The rodent forestomach has a different anatomy than the human stomach and may represent an unusually sensitive assay of the carcinogenicity of BaP by the oral route. The absence of tumors at sites other than in the forestomach raises questions as to the significance of these findings to the risks to humans from ingestion of BaP.

- Assuming that the positive findings indeed provide a basis for extrapolation to humans, although dosing route may be significant, we have assumed that absorbed dose by inhalation or ingestion has the same effect.

- A vital consideration in the extrapolation of risks associated with PAHs in general, and BaP specifically, is the existence of metabolic differences, both qualitatively and quantitatively, between humans and other species.

The degree of individual genetic variation of some activating enzymes is suspected to be a key factor in an organism's susceptibility to PAH-induced carcinogenesis. The highly heterogeneous nature of human populations exposed to BaP introduces a confounding factor in reliably predicting excess cancer incidences due to BaP exposure.

- Both animal studies, and especially the study by Neal and Rigdon (1967), were of less than lifetime duration. Doses were administered for about half the duration of the experiment in both studies. In order to adjust animal dose

TABLE 5-64. ESTIMATED LIFETIME EXCESS PROBABILITY OF CANCER TO HUMANS DUE TO BENZO[a]PYRENE INGESTION AT VARIOUS EXPOSURE LEVELS BASED ON THREE EXTRAPOLATION MODELS[a]

	Estimated Lifetime Excess Probability of Cancer at Indicated Exposure Level					
Exposure Level (μg/day)	0.001	0.01	0.1	1	10	100
Extrapolation Model						
Linear Model						
Neal and Rigdon data	1×10^{-6}	1×10^{-5}	1×10^{-4}	1×10^{-3}	1×10^{-2}	9.2×10^{-2}
Fedorenko and Yanysheva data	1.1×10^{-5}	1.1×10^{-4}	1.1×10^{-3}	1.1×10^{-2}	1.1×10^{-1}	6.7×10^{-1}
Log-probit Model						
Neal and Rigdon data	1×10^{-9}	1×10^{-7}	1.4×10^{-5}	7.5×10^{-4}	1.5×10^{-2}	1.2×10^{-1}
Fedorenko and Yanysheva data	3×10^{-7}	2.6×10^{-5}	1.2×10^{-3}	2.1×10^{-2}	1.5×10^{-1}	4.8×10^{-1}
Multi-stage Model						
Neal and Rigdon data	1.7×10^{-6}	1.7×10^{-5}	1.7×10^{-4}	1.7×10^{-3}	1.6×10^{-2}	1.5×10^{-1}
Fedorenko and Yanysheva data	7×10^{-6}	7×10^{-5}	6.8×10^{-4}	6.8×10^{-3}	6.5×10^{-2}	4.8×10^{-1}

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[a] The lifetime excess probability of cancer represents the increase in probability of cancer over the normal background incidence, assuming that an individual is continuously exposed to BaP at the indicated daily intake over a 70-year lifetime. There is considerable variation in the estimated risk due to uncertainty introduced by the use of laboratory rodent data, by the conversion to equivalent human dosage, and by the application of hypothetical dose-response curves. In view of several conservative assumptions that were utilized, it is likely that these predictions overestimate the actual risk to humans.

schedule to an equivalent daily lifetime human dose, two assumptions were utilized as recommended by the USEPA (1979). First it was assumed that the response would be the same if the total dose was distributed evenly over the entire experimental lifetime of the animal. Second, it was assumed that a short-duration study attributable to either early mortality or early termination leads to an overestimation of the dose required to produce the observed effect. The average daily dose for the Neal and Rigdon data was adjusted to reflect the actual experimental lifetime versus the theoretical lifetime of the experimental animal. In treating the Neal and Rigdon data, this adjustment lead to a marked reduction (∿1/40) in the effective daily dose level.

- Slight differences in dose-effect relationship between the Neal and Rigdon (1967) and the Fedorenko and Yanysheva (1966) studies which were primarily attributed to the difference in mode of administration (diet vs. gavage, respectively). These may also have been caused by differences in dosing schedules.
- It appears that the total effective dose for the Neal and Rigdon study is probably not a simple product of daily dose times duration but, in fact, may be a more complex power function in which the dose rate has a greater influence on response than treatment duration.
- Development of PAH-induced tumors can be altered by components in the diet, exposure to inducers or inhibitors of microsomal enzymes, as well as cocarcinogenic effects such as from substances present in cigarette smoke.

Keeping these qualifiers in mind, the risks shown in Table 5-64 were combined with the known BaP exposure levels (Table 5-63) to estimate per capita lifetime risks and incidence (excess cancers/year) for BaP exposure routes as shown in Table 5-65.

The highest estimated carcinogenic risks appear to be associated with the subpopulation that smokes. In the general population, the highest estimated risks are associated with exposure to BaP in the diet (e.g., charcoal-broiled meats and fish). Inhalation exposure at the upper limit of the reported concentration ranges in urban areas are higher but are based on limited monitoring data. In contrast, continuous lifetime consumption of BaP-contaminated drinking water at typical concentration levels presents the lowest potential carcinogenic risks.

TABLE 5-65. ESTIMATED RANGES OF CARCINOGENIC RISK TO HUMANS DUE TO BENZO[a]PYRENE EXPOSURE FOR VARIOUS ROUTES

Route	Average Lifetime BaP Exposure (μg/day)	Size of Exposed Population	Estimated Lifetime Excess Probability of Cancer[a]	Estimated Incidence (excess cancers/year)
Typical Diet	0.05	221×10^6	4×10^{-6} to 6×10^{-4}	13 - 1,900
Drinking Water	0.0006	221×10^6	1×10^{-10} to 7×10^{-6}	<< 1 - 22
Ambient Air - Urban	0.02 - 2	166×10^6	6×10^{-7} to 4×10^{-2}	1 - 95,000
- Rural	0.0002 0.2	55×10^6	3×10^{-11} to 3×10^{-3}	<< 1 - 2,400
Smoking	0.6[b]	54×10^6[b]	3×10^{-4} to 1×10^{-2}	230 - 7,700

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[a] A range of probability is given, based on several different dose-response extrapolation models. The lifetime excess probability of cancer represents the increase in probability of cancer over the normal background incidence, assuming that an individual is continuously exposed to BaP at the indicated daily intake over a 70-year lifetime. There is considerable variation in the estimated risk due to uncertainty introduced by the use of laboratory rodent data, by the conversion to equivalent human dosage, and by the application of hypothetical dose-response curves. In view of several conservative assumptions that were utilized, it is likely that these predictions overestimate the actual risk to humans.

[b] It was assumed that the total population of smokers (54 million) smoked on average 25 cigarettes per day. From 25% to 30% of smokers consume more than 25 cigarettes per day and consequently may receive a higher daily exposure.

With respect to other PAHs included in this group, data were inadequate for quantitative risk assessment purposes. Benz[a]anthracene and dibenz[a,h]anthracene are both carcinogenic in mice by the oral route. They are also complete carcinogens for mouse skin as are benzo[b]-fluoranthene and indeno[1,2,3-c,d]pyrene. Benzo[g,h,i]perylene is a co-carcinogen with BaP and benz[a]anthracene, benzo[b]fluoranthene, chrysene and indeno[1,2,3-c,d]pyrene are all initiators of skin carcinogenesis in mice. No carcinogenicity data were found for acenaphthylene.

5.5.3.2 Non-Carcinogenic Risks

The major focus of studies with compounds in the benzo[a]pyrene group has been their potential for inducing carcinogenic effects. Little information is available on other potential risks to humans from exposure to these compounds (see Section 5.3.1). BaP has been shown to be the most active mutagen of the group, inducing in vivo chromosomal aberrations and sister chromatid exchange in hamsters. Mixed mutagenic responses have been noted with other compounds in this group. Because of its mutagenicity, BaP exposure could also be expected to contribute to the genetic burden of a population, but since extrapolation procedures for genetic risks have not been well established, a quantitative risk assessment for these kinds of health hazards is not presently feasible.

BaP appears to exert little effect on the developing embryo; data were unavailable for the other compounds in this group.

Little information is available on other possible toxic effects associated with exposure to these compounds. However, in assessing the risks to humans associated with exposure to these PAHs, one should not overlook possible augmentation of effects through synergistic or co-carcinogenic mechanisms. Current understanding of the co-carcinogenesis process is not sufficiently adequate to allow estimation of human risk at this time.

5.5.4 Risk to Biota

Data on acute and chronic effects of PAHs are limited and water quality criteria have not been set (USEPA 1980). The only laboratory toxicity test for any of these compounds indicates that 1.0 µg/l of benz[a]anthracene caused 87% mortality in bluegill sunfish in six months. Concentrations in this general range (0.2 - 20 µg/l) have also been found to stimulate growth in some organisms including baceria and freshwater algae. Several field studies have attributed certain tumors found in benthic marine fish to BaP from hydrocarbon-contaminated sediments, but the concentrations of BaP causing these effects were not known.

Monitoring data at detectable levels were very scarce, but those which were available indicate that benz[a]anthracene and benzo[k]fluoranthene have been found in surface water at concentrations from 1 - 1500 µg/l. Sediment concentration data were somewhat more extensive and indicated that all of the eight PAHs in this group have been detected in concentrations exceeding 1.0 µg/kg. Maximum sediment concentrations for this group ranged from 35.8 µg/kg (indeno[1,2,3-c,d]pyrene) to 2600 µg/kg (dibenz[a,h]anthracene). Uptake of PAHs from sediments by benthic organisms is believed to occur, but the extent of bioavailability of sediment-bound PAHs is not known at this time. Although no data were found attributing specific sediment PAH concentrations to toxic effects, it is believed that PAHs from hydrocarbon-contaminated sediments have caused liver tumors in fish. Many of the higher sediment monitoring observations were found in Puget Sound, the area in which liver tumors in fish have been observed. PAH sediment levels, therefore, which are often found near industrialized areas, may pose some risk to aquatic biota.

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APPENDIX A

NOTE 1:

Fireplaces

Emissions were calculated from emission factors (see Table A-1) that represented the average of three tests (EPA 1980b). The total mass of wood burned in fireplaces in 1976, 2.7×10^6 kkg (EPA, 1980a) was extrapolated to 1977, based upon the number of housing units in each region of the country, the percentage of those housing units with fireplaces an average consumption of 98.3 kg/housing unit (EPA 1980a). These assumptions led to the estimate that 2.9×10^6 kkg of wood were burned in 1977.

NOTE 2:

Carbon Black

An estimated 1.0×10^6 kkg of carbon black were produced in 1977 (SRI, 1979). The emission factors in Table A-1 (Serth and Hughes, 1980) were used to estimate PAH emissions, although the resulting estimates are limited by the fact that testing was performed upstream of an emissions control device (burner).

An estimate of PAH production associated with carbon black manufacture is presented in Table A-3.

Tire Wear

A crude estimate of PAH emission from tire wear associated with carbon black (see Table A-4) was developed from the following data:

- 365 da/yr.
- 7.4×10^6 bbl/da gasoline consumption (Oil and Gas Journal 1979).
- 14.7 miles/gal average (EPA 1975).
- 42 gal/bbl.
- 0.19 g/vehicle-mile is airborne particulates and 0.15 g/vehicle-mile is deposited on road surface (EPA 1979c).
- Rubber composed of 33% carbon black (SRI 1979).
- Average PAH composition of carbon black in Table A-3 (Locati *et al*. 1979).

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NOTE 3:

Motor Vehicles

Motor vehicle PAH emissions were calculated by using the emission factors in Table A-1 (Hangebrauck, 1967), and estimated 2.7×10^{15} meters/yr travelled (see note on tire wear fcr estimation of vehicle miles travelled), and an assumed emission reduction of two thirds. this emission reduction was based upon the following information:

Fraction of Automobile		
Population	Control	Present Reduction
0.32	catalytic Conventer	99
0.58	Engine Modification	65
0.10	None	0

The automobile population is from EPA (1978b), and the percent reductions are based on ranges mentioned in that document.

The concentrations of PAHs in used crank case oil are shown in Table A-5. Releases to sewers and landfills were assumed to account totally for 2×10^9 ℓ of oil disposed of by the public (Tanacredi 1977); however, this figure does not take into account used oil that is recycled.

NOTE 4:

a. Oil:

Water figure based on 3.6×10^7 ℓ of various oils - crude (36%), diesel (18%), fuel (42%), waste (2%), lube (0.3%) other (1.7%) - spilled in navigable waters in 1978 (U.S. Coast Guard, 1980).

Land figure based on 5.1×10^5 ℓ of crude oil spilled in 1978 by common carrier (23%), private carrier (22%), rail (6%), and "other" (49%) (U.S. Dept. of Transportation 1980). Average oil Density = 0.85.

b. Gasoline:

Water - 1.1. $\times 10^7$ ℓ spilled: aviation/automobile gasoline (98%) and natural (Casinghead) Gasoline (2%) (U.S. Coast Guard 1980).

Land - 3.7×10^6 ℓ spilled: common carrier (53%), private carrier (47%), rail (0.8%), "other" (<0.01%) U.S. Dept. of Transportation 1980). Gasoline density: 0.73.

Table A-1. Emission Factors

	Residential Coal Combustion (g/kg)	Fireplaces (g/kg)	Primary and Auxiliary Wood Heating (g/kg)	Cigarettes (μg/cig)	Coal Refuse Piles (kg/kg) POM	Forest Fires (ng/g) (dry fuel)	Carbon Black μg/kg	Gasoline (ng/meter)
Acenaphthene	0.039	0.0012	0.0076					
Acenaphthylene		0.010	0.057				800	
Anthracene[a]	0.008	0.010	0.076	0.17	0.1	2,500	35	4.3
Benzo[a]anthracene	0.002	0.0008	0.0071	0.02		3,100	4.5	
Benzo[b]fluoranthene[c]	0.002	0.0008	0.0058		0.01	1,300	15	
Benzo[k]fluoranthene	0.002	0.0008	0.0058		0.01	1,300	15	
Benzo[ghi]perylene		0.0009	0.0053			2,500	12	47
Benzo[a]pyrene[d]	0.0015	0.0008	0.0040	0.01	0.005	740		11.5
Chrysene[b]	0.002	0.0008	0.0071	0.02		3,100	4.5	
Dibenzo[a,h]anthracene[e]	0.003	0.0001	0.0007		<0.001			
Fluroanthene	0.005	0.0028	0.019	0.01	0.05	5,500	60	75
Fluorene	0.026	0.0047	0.016					
Indeno[1,2,3-cd]pyrene	0.002	ND	ND	0.006	<0.001	1,700	<2	
Naphthalene	0.15	0.0403	0.25	3				
Phenanthrene	0.008	0.010	0.076	0.36	0.1	2,500	35	30
Pyrene	0.005	0.0028	0.016	0.16	0.05	4,600	500	110

a) Reported as anthracene/phenanthrene, assumed equal division between them.
b) Reported as chrysene/benzo[a]anthracene, assumed equal division between them.
c) Reported as benzo fluoranthenes, assumed divided solely between benzo[b]fluoranthene and benzo[k]fluoranthene.
d) Reported as benzopyrene(s) and perylene, assumed to be 50% benzo[a]pyrene.
e) Reported as dibenzanthracene, assumed to be solely dibenzo[a,h]anthracene.

Sources listed in Section 5.1 text.

Table A-2. Fireplace Population

Region	Number of Housing Units (1977)	Percentage w/Fireplace	Fireplaces
Northeast	17,707,000	47	8,300,000
North Central	21,181,000	33	7,000,000
South	26,422,000	29	7,700,000
West	15,406,000	46	7,100,000
Total			30,100,000

Sources: Census 1979 and EPA 1980a

Table A-3. PAH Associated with Carbon Black (μg/g)[a]

PAH \ Carbon Black Type	Vulcan J	Regal 300	330 HAF	660 GPF	339	Avg	Contained in Carbon Black (kkg)
Anthracene[b]	0.5	ND	0.05	ND	1	0.3	0.5
Benzofluoranthenes[c]	10	ND	<0.9	4	7	4	6
Benzo[ghi]perylene	166	16	25	41	164	82	100
Benzopyrenes	20	1	3	8	32	17	30
Fluoranthene	68	9	10	13	52	30	50
Indenopyrene	24	1	0.3	7	35	13	20
Phenanthrene	0.5	ND	0.05	ND	1	0.3	0.5
Pyrene	314	58	47	52	207	140	200

a) Based 1.6 x 10^6 kkg carbon black production (SRI 1979).
b) Reported as anthracene/phenanthrene, assumed equal division among them.
c) Excluding benzo[ghi]fluoranthene, reported separately.

Source: Locati *et al*. 1979

Table A-4. PAH Releases from Tire Wear (kkg)[a]

	Airborne		Sedimentary or directly transferred to Roadway	Total
	Initial	Reentrained		
Anthracene				
Benzofluoranthenes				
Benzo[ghi]perylene	1	7	7	20
Benzopyrenes	0.3	1	1	2
Fluoranthene	0.5	3	2	6
Indenopyrene	0.2	1	1	2
Phenanthrene				
Pyrene	2	10	10	20

See Appendix text for calculations and sources

a) Blanks indicate <1 kkg/yr. For all entries, totals may not add due to rounding.

Table A-5. Concentrations of PAHs in Used Crankcase Oils (mg/l)

Anthracene	0.3
Benzo[a]anthracene	0.9
Benzo[k]fluoranthene	1.4
Benzo[ghi]perylene	1.7
Benzo[a]pyrene	0.4
Chrysene	1.2
Fluoranthene	4.4
Fluorene	1.5
Phenanthrene	7.8
Pyrene	6.7

Source: Peake and Parker 1980.

Table A-6. Municipal Incinerators Release Factors (μg/kg refuse)

	Air[a]	Land	Water[b]
Benzo[a]anthracene[c]	1.5	18	0.08
Benzo[b]fluoranthene[d]	0.5	21	0.01
Benzo[k]fluoranthene[d]	0.5	21	0.01
Benzo[ghi]perylene	1.8	10	0.007
Benzo[a]pyrene[e]	0.04	16	0.016
Chrysene[c]	1.5	18	0.08
Fluoranthene	2.5	12	0.14
Indeno[1,2,3-cd]pyrene	0.77	<2.1	<0.002

a) after scrubber
b) taken as one half reported benzo[a]anthracene + chrysene emissions
c) taken as one third of benzo[b+k+j]fluoranthene emissions
d) taken as one half of benzo[a+e]pyrene emissions
e) scrubber water

Source: Davies 1976.

Table A-7. Emissions of PAHs from Coal-fired Plants and Intermediate/small Oil-fired Units, μg/10^9 J Fuel

Type of Unit	Benzo[a]-pyrene	Pyrene	Benzo[ghi]-perylene	Phenan-threne	Fluoran-thene
Pulverized coal (vertically-fired, dry-bottom furnace)	18 - 123	70 - 218	79		80 - 389
Pulverized coal (front-wall-fired, dry-bottom furnace)	16 - 20	152 - 190	13	190	12 - 152
Pulverized coal (tangentially-fired, dry-bottom furnace)	123	133	142	30	370
Pulverized coal (opposed-, downward inclined burners; wet bottom furnace)	20 - 133	37 - 114	142 - 1,042		52 - 199
Crushed coal (cyclone-fired, wet-bottom furnace)	72 - 351	237 - 1,706	34 - 341		42 - 104
Spreader stoker (traveling grate)	<14 - 23	20 - 56			20 - 56
Oil-fired:					
Steam atomized	<19 - 45	46 - 284		1,700	53 - 256
Low pressure air atomized	853	5,780	285	3,320	1,800
Pressure atomized	<38 - <57	14 - 1,700		8,440	72 - 4,470
Vaporized	<95	1,140			14,200

NOTE: Blanks indicate data not available.

Source: Hangebrauck et al. 1967.

Table A-8. Coke-Oven Tar Produced in the United States, Used by Producers, and Sold in 1978 by State (Thousand Liters)

	Produced		Used by Producers			Sold for Refining into Tar Products			
							Value		
State	Total	L/kkg of Coal Coked	For Refinery or Topping	As Fuel	Other	Quantity	Thousand Dollars	Average Per Liter	On-hand Dec. 31
Alabama	130,000	27	a	-	-	130,000	$11,970	$0.09	9,700
Calif., Colo., Utah	130,000	35	-	-	-	130,000	11,905	0.09	13,000
Illinois	59,000	25	-	-	-	60,000	5,593	0.09	5,700
Indiana	350,000	33	a	a	a	120,000	12,840	0.11	21,000
Ken., Mo., Tenn., Tex.	34,000	25	-	a	-	31,000	3,021	0.10	2,700
Maryland, New York	170,000	32	-	a	-	120,000	12,911	0.11	25,000
Michigan	a	a	-	-	-	a	a	a	a
Minnesota, Wisconsin	20,000	23	-	a	-	19,000	1,938	0.10	a
Ohio	330,000	31	-	170,000	a	180,000	17,529	0.10	27,000
Pennsylvania	580,000	35	a	a	a	280,000	29,425	0.11	55,000
Virginia, West Virginia	a	a	a	-	-	a	a	a	5,700
Undistributed	240,000	30	500,000	190,000	32,000	150,000	12,904	0.09	14,000
Total (1978)[b]	2,100,000	44	500,000	360,000	32,000	1,200,000	120,036	0.10	180,000
At Merchant Plants	89,000	34	c	-	-	89,000	8,986	0.10	4,900
At Furnace Plants	2,000,000	44	500,000	360,000	32,000	1,100,000	111,050	0.10	170,000
Total (1977)[b]	2,200,000	32	570,000	550,000	38,000	1,100,000	106,728	0.10	160,000

a) Included with "Undistributed" to avoid disclosing individual company data.
b) Data may not add to totals shown due to independent rounding.
c) Included with "Furnace Plants" to avoid disclosing individual company data.

Source: DOE 1979.

Table A-9. Concentrations of Various PAHs in Coal and Coal Tar Derivatives (mg/kg)[a]

	Coal	Coal Tar	Coal Tar Pitch	Creosote Oil
Acenaphthene		10,000[b]		40,000[c]
Anthracene		9,000[b]		20,000[c]
Phenanthene		30,000[d]		100,000[c]
Benzo[a]anthracene		<0.007[d]	<10[d]	<3[d]
Benzo[a]pyrene	0.7	30[d]	10[d]	neg[d,e]
Chrysene		4,000[d]	<10[d]	<1[d]
Fluoranthene		6,000[b]		40,000[c]
Fluorene		10,000[b]		30,000[c]
Naphthalene		90,000[f]		200,000[c]
Pyrene		3,000[g]		30,000[c]

a) All numbers rounded to one significant figure; blank spaces = data not available.
b) Sources: Rhodes 1954; Lowry 1945 (averaging of data).
c) Source: Stasse 1954; as cited in Weiler 1963.
d) Source: IARC 1973.
e) Less than 0.01 ppm.
f) Source: Rhodes 1954; and Lowry 1945.
g) Source: Rhodes, 1954.

Table A-10. PAH Wastewater Discharge: By-Product Cokemaking[a]

	Discharge Factors (kg/kkg coke)			
	Ammonia Liquor	Cooler Blowdown	Benzol Plant	(kkg) Total Discharge
Acenaphthylene	0.00073	0.000097	0.000129	40
Benzo[a]anthracene	0.000006	0.000032	0.000125	7
Benzo[a]pyrene	0.000032	0.000024	ND	3
Chrysene	0.000045	0.000018	0.000155	10
Fluoranthene	0.000196	0.000323	0.000189	30
Fluorene	0.000145	0.000048	0.000049	10
Naphthalene	0.00395	0.0115	0.00341	800
Pyrene	0.000393	0.000026	0.000109	20

a) Based on the total of three factors and a 1978 coke production of 44.5 x 10^6 kkg (DOE 1979). Distribution: 33%-direct, 25%-POTWs, 2%-deep well, 40% quenching (20% land, 20% air) (EPA 1979d).

Source: EPA 1979d.

Table A-11. Concentration of Select PAHs in Petroleum Products, mg/kg

	Crude Oil	Gasoline	Kerosene	Petroleum Asphalt	Diesel Fuel	Number 2 Heating Oil
Acenaphthene	ND[a]					
Acenaphthylene	400					
Anthracene	trace	3	0.4		3	4
Benzo(a)anthracene	trace	3	<0.1	0.04	0.1	0.04
Benzo(b)fluoranthene	<5					
Benzo(k)fluoranthene	<5					
Benzo(ghi)perylene	.02	2	<0.1		0.03	0.03
Benzo(a)pyrene	1	2	0.01	0.01	0.07	0.03
Chrysene	<100	2	ND	0.02	0.5	0.6
Dibenzo(a,b)anthracene						
Fluoranthene	100	7	0.09		0.5	2
Fluorene	200					
Indeno[1,2,3-cd]pyrene						
Naphthalene	1,000					
Phenanthrene	100		ND		ND	ND
Pyrene	100	5	0.2		0.4	1

a) ND means not detected.

Source: Guerin 1978; Guerin et al. 1978; EPA 1979f

Table A-12. Emissions of PAHs from Petroleum Refining

Process	Emissions, µg/100 m^3[a]	
	Benzo(a)-pyrene	Benzo(ghi)-perylene
Straight run distillation	0.015	0.11
Pyrolysis	0.30	0.170
Asphalt production	0.74	1.58
Petroleum coke production	25.5	0.4
Petroleum products purification	0.024	–[b]

a) Mean values of multiple samples.

b) Not available.

Source: Samedov and Kurbanov 1971, as cited in EPA 1979h.

Table A-13. Emissions of PAHs from Catalyst Regeneration in Petroleum Cracking, μg/m^3 Oil Charges

Type of Unit	Benzo(a) pyrene	Pyrene	Benzo(ghi)-perylene	Anthracene	Phenanthrene	Fluoranthene
FCC:[a]						
Regenerator outlet	0.7 - 73	6.4 - 4,450	24 - 67		63,560	7.0 - 3,180
Carbon monoxide boiler outlet	1.7 - 3.4	3.9 - 26	8.8	330		3.2 - 13
HCC:[b]						
Regenerator outlet	32,600 - 36,700	20,700 - 20,800	47,700 - 60,400	146 - 318	3,340 - 4,600	1,320 - 1,810
TCC:[c]						
Air lift, regenerator outlet	8,900 - 19,100	21,000 - 41,300	7,000 - 11,450	1,640 - 1,685	52,500 - 56,000	1,685 - 4,610
TCC:						
Brucket lift, regenerator outlet	5	46 - 57				9.17

NOTE: Blanks indicate data not available.

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a) Fluid catalytic cracking.
b) Houdriflow catalytic cracking.
c) Thermofor catalytic cracking.

Source: Hangebrauck, *et al*. 1967.

APPENDIX B. APPLICATION OF THE AIR-TO-SURFACE PATHWAY EVALUATION METHOD FOR POLYNUCLEAR AROMATIC HYDROCARBONS

A method has been developed for estimating airborne toxicant deposition rates (air-to-surface pathway evaluation method, Arthur D. Little, Inc., 1981). The method accounts for both wet and dry deposition to land and water surfaces. When deposition to a watershed or other land area is estimated, this can be interpreted as an upper bound on the chemical loading to an associated surface water body resulting from air deposition, since only a fraction of the mass deposited on land surfaces will be delivered to the water body. The air-to-surface pathway evaluation method accounts for the partitioning of an airborne contaminant between adsorbed and vapor phases, with differing deposition rates inferred for the separate phases. It relies on fundamental physicochemical properties and is designed to use available data, while filling in data gaps with estimated values of various parameters. The evaluation method has been applied to naphthalene, anthracene, and benzo[a]pyrene. Each of the three PAHs modeled has an atmospheric chemical degradation of roughly 0.1 hr.$^{-1}$ leading to half-lives of 5-10 hours (Radding _et al_. 1976). Under typical meteorologic conditions this corresponds roughly to the travel time across major urban areas. Since urban areas also would be expected to have much greater emission densities than rural areas, significant urban/rural differences in air concentrations of PAHs are expected, and indeed observed. These factors suggest that deposition rates under urban and rural conditions should be considered separately.

One of the most important chemical properties influencing air-to-surface transfer is the vapor pressure. The vapor pressure affects the partitioning of airborne contaminants between vapor and adsorbed phases. The deposition rate is typically much greater for the adsorbed fraction of the airborne contaminant. The effect of vapor pressure is expressed by equation (5) of the Arthur D. Little report (1981):

$$\phi = \frac{.165\theta}{P_o + .165}$$

where

ϕ is the adsorbed fraction of the total airborne mass,

θ is the available aerosol surface area $\frac{cm^2}{cm^3}$ and

P_o is the saturation vapor pressure of the contaminant at ambient temperature (torr)

The available aerosol surface area is typically greater in urban areas where concentrations of total suspended particulates are higher

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than in rural areas. In the plume from a combustion source, the aerosol surface area is even higher than typically found in urban air. Application of the above equation results in aerosol partitioning for the three PAHs as shown below:

PAH	Adsorbed Fraction of Total Airborne Mass		
	Rural	Urban	Near Combustion Sources
Naphthalene	7×10^{-6}	3×10^{-4}	0.02
Anthracene	0.002	0.06	0.97
Benzo[a]pyrene	.99	1.00	1.00

Benzo[a]pyrene is strongly partitioned with the aerosol phase, regardless of ambient conditions, while at the other extreme naphthalene exists primarily as a vapor in the atmosphere. Anthracene exhibits intermediate properties, and the adsorbed fraction is sensitive to ambient conditions.

The dry deposition flux is proportional to the dry deposition velocity, V_d, i.e.,

$$\text{Dry Flux} = V_d \, C_{air}$$

where

C_{air} is the ground-level air concentration.

The dry deposition velocity with respect to the total airborne contaminant is calculated as the (mass) weighted average of the deposition velocity for the vapor and sorbed fractions, i.e.,:

$$V_d = V_{d,s}\phi + V_{d,v}\,(1-\phi)$$ (Eq. 6 of Arthur D. Little 1981)

According to the air-to-surface pathway method (Arthur D. Little 1981), the respective dry deposition velocities are given below:

PAH	Dry Deposition Velocity				
	$V_{d,v}$ (cm/sec)	$V_{d,s}$ (cm/sec)	Rural	Urban	Combustion Source
Naphthalene	0.04	1	0.04	0.04	0.06
Anthracene	0.02	1	0.02	0.08	1.00
Benzo[a]pyrene	0.02	1	1.00	1.00	1.00

Wet deposition is controlled by the precipitation scavenging ratio, r, which expresses the ratio of pollutant concentration in precipitation (ng/l) to pollutant concentration in air ($\mu g/m^3$). The scavenging ratio is calculated by contributions from the vapor and sorbed fractions, i.e.:

$$r = r_s\ (\phi) + r_v\ (1-\phi)$$

where r, r_s and r_v are scavenging ratios for the total airborne mass, the sorbed contaminant, and the vapor phase, respectively. Once the scavenging ratio is known, the wet deposition flux is given by wet flux = rRC_{air}, where R is the rainfall rate. These parameter values have been estimated, as shown below:

	Precipitation Scavenging Ratios				
			r		
PAH	r_s*	r_v	Rural	Urban	Combustion Sources
Naphthalene	$6x10^4$	53	53	71	$2.5x10^3$
Anthracene	$6x10^4$	14	130	$3.6x10^3$	$1.2x10^5$
Benzo[a]pyrene	$6x10^4$	$5.4x10^4$	$6x10^4$	$6x10^4$	$1.2x10^5$

For the purpose of further analysis a generic urban environment has been modeled on the basis of characteristics of Philadelphia and Cleveland. The average wind speed is 10.25 kts (5.3 m/s) and the annual rainfall is 1.0 m/yr. The urban area is 100 mi^2 ($2.6 \times 10^8\ m^2$).

The generic rural area is defined as a volume of air within which an associated urban source would contribute to rural concentrations. The size of the area is constrained by the half-life in air, such that the concentration is typical of an area significantly affected by the urban source. At a half-life of 5 hours, and typical wind speed of 5.3 m/sec., naphthalene contamination from an urban area could be significant over an area of $10^{10}\ m^2$ (roughly 40,000 square miles).

Median observed ambient concentrations for three PAHs in urban and rural areas are presented below (White and Vanderslice 1980):

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*Tabulated r_s values apply for rural and urban conditions. Near a combustion source the adsorbed phase is expected to be associated with larger particles resulting in a scavenging ratio, $r_s = 1.2x10^5$.

PAH	Median Observed Concentration (ug/m^3) Rural	Urban
Naphthalene	$7x10^{-5}$	$5x10^{-4}$
Anthracene	$1x10^{-3}$	$8x10^{-3}$
Benzo[a]pyrene	$1x10^{-3}$	$1x10^{-2}$

Using the pathway evaluation method we have estimated the emission that would result in a specific ambient concentration, given the degradation rate, deposition velocity, rainfall rate, and precipitation scavenging ratio. Applying the equation for wet flux and dry flux we also estimated the amount deposited in the generic rural and urban study areas. Then, by comparison of the deposition rates with emission rates, we estimated the fraction of total atmospheric emissions of each of the three PAHs which would be deposited within the urban and rural study areas. These results are shown below:

	Deposition Rate					
PAH	% of Emissions Dry Deposited Rural	Urban*	% of Emissions Wet Deposited Rural	Urban	% Deposited Rural	Urban
Naphthalene	2	2-3	<1	<1	2	2-3
Anthracene	1	4-19	<1	1-7	1	5-26
Benzo[a]pyrene	22	19	4	4-7	26	23-26

From the results above, it is apparent that a very small fraction of atmospheric emissions of naphthalene are deposited on land or water surfaces. The fraction of air emitted anthracene that is eventually deposited is uncertain, but could be fairly large percentage. Approximately one-fourth of all benzo[a]pyrene emitted into the atmosphere will eventually be deposited on land and water surfaces, where it would contribute to the contamination of surface runoff and surface water bodies.

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*The range for urban areas reflects alternative assumptions that the chemical has equilibrated with ambient aerosol or remains associated with particulates in the plume from the combustion zone.

APPENDIX
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